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Vol. XXXVI DECEMBER, 1926 No. 12

THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
DEVOTED TO DISEASES OF THE

EAR - NOSE - THROAT

FOUNDED IN 1896 BY

DR. M. A. GOLDSTEIN

Managing Editor and Publisher.

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Subscription, $6.00 per Annum, in Advance.

Foreign Subscription. 35 Shillings per Annum. Post Free.
Single Copies, 75 cents.

PUBLISHED BY THE LARYNGOSCOPE CO.

3858 Westminster Place, St. Louis, Mo. U. S. A.

FOREIGN OFFICE, BAILLIERE, TINDALL & COX,
8 HENRIETTA ST., STRAND, LONDON, ENG.

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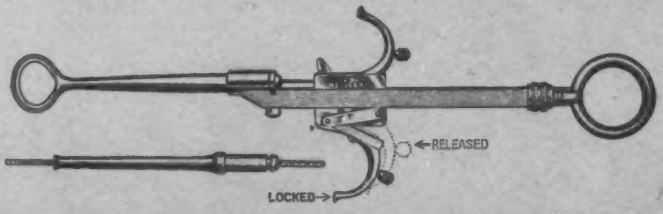

THE
LARYNGOSCOPE.

VOL. XXXVI DECEMBER, 1926 No. 12

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

PAPILLEDEMA FROM THE VIEWPOINT OF THE OTOLOGIST.*

DR. EDWARD B. DENCH, New York.

I wish to preface the few remarks which I make by the statement that I shall discuss the changes occurring in optic papilledema simply from the viewpoint of the otologist. Unfortunately, I know little about the microscopic anatomy of the various conditions of the fundus, neither do I know the finer physical differentiations between the simple edema of the disc and an edema combined with an inflammation of the filaments of the optic nerve; in other words, an optic neuritis or neuroetinitis as distinguished from a simple swelling of the optic disc. Unfortunately, I have not used the ophthalmoscope myself for many years and have always depended upon my ophthalmological confreres to give me the fundus findings in all cases. This will explain my ignorance of the more technical terms which are used concerning the various conditions.

In the first place changes in the fundus oculi may occur as the result of any intracranial condition which gives rise to increased intracranial pressure. We are concerned tonight simply with those cases in which the intracranial pressure is increased by some otitic condition. Although it is my impression that one or two cases have been reported in which a simple inflammation of the mastoid without any intracranial complication was responsible for a papilledema, or at least for some abnormality in the optic disc, I myself have never seen such a condition. Perhaps if the fundus oculi were examined

*Read before the Section on Otology, New York Academy of Medicine, Feb. 12, 1926. Symposium on Papilledema.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication May 13, 1926.

in each case of mastoid involvement, and this I think would be a very wise and valuable procedure, it is possible that changes in the ocular fundus might be found. In my own experience, however, I have never observed a case of papilledema complicating a middle ear condition unless some intracranial condition were present. It should be stated further that any of the intracranial complications of middle ear suppuration may be accompanied by certain changes in the optic disc. These changes may vary from a slight engorgement and tortuousity of the veins to an actual swelling of the head of the optic nerve, and blurring of the margins of the disc. Under certain conditions an actual neuritis of the optic nerve takes place.

The first change, that is, an increased tortuousity and dilation of the veins with blurring of the margins of the disc, is the result of increased intracranial pressure. Such a fundus change may occur as a result of any intracranial complication from any cause, and all of the intracranial complications of middle ear suppuration may cause these changes to a varying degree. The intracranial middle ear complication least likely to cause a papilledema is an extradural abscess; that is, a collection of pus between the dura and the calvarium. This, the simplest and least dangerous of the intracranial complications, may cause changes in the appearance of the optic disc, but rather infrequently. It should be understood that while as a rule if these optic disc changes are unilateral they usually occur on the side of the ear lesion, this rule is by no means absolute. I have seen cases in which the optic disc of the affected side was perfectly normal, and the opposite disc showed a marked papilledema. This applies, of course, not only to cases of extradural abscess, the simplest of intracranial complications of middle ear suppuration, but to all of the other intracranial complications of this disease as well. Sinus thrombosis, otitic meningitis, and both cerebral and cerebellar abscess are all prone to produce changes in the optic disc.

Without going into the exact figures, I should say that optic disc changes were more constant in meningitis than in any of the other intracranial complications of otitic origin. They occur, very frequently, however, in brain abscess, both cerebral and cerebellar abscesses and also in cases of sinus thrombosis. In the cases of brain abscess and of meningitis, the general condition of the patient is frequently so desperate that the condition of the fundus oculi is simply sought as a corroborative diagnostic measure. In cases of sinus thrombosis, however, the general condition of the patient is not so serious and the fact that these changes have taken place in the optic disc is important not only because they constitute an important diagnostic feature of an intracranial condition, but also because the

effect of the condition on the subsequent vision of the patient has to be considered. I think that I can safely say in the majority of cases where we have had a sinus thrombosis necessitating either ligation or excision of the internal jugular vein that certain changes have been present in the optic disc on one or both sides. If the condition is allowed to persist there is danger of serious impairment of vision.

In these cases repeated lumbar punctures have, at my hands, been entirely successful in relieving the increased intracranial pressure and in all of these cases the vision became normal. Repeated lumbar puncture, then, has in my hands been the best method in preserving vision in cases of optic nerve changes following intracranial complications. While, of course, in brain abscess, evacuation of the abscess ordinarily relieves the intracranial pressure to such a degree that optic nerve changes retrogress rapidly, in their persistence lumbar puncture is of undoubted value—and the same applies to cases of meningitis.

Neoplasms either above or below the tentorium may give rise to certain disturbances of the eighth nerve, bringing these cases under the observance of the otologist. Here the association of certain aural conditions with signs of intracranial pressure are of great value in confirming the diagnosis in doubtful cases. The same applies to certain aural conditions following constitutional diseases, such as lues, diabetes, arteriosclerosis and others.

The point that it seems to me more important to bring out is that even slight changes in intracranial pressure are sufficient to cause marked changes in the ocular fundus. In one case reported by me a number of years ago in which following an acute middle ear suppuration the Gasserian ganglion was freed from dural adhesions, I introduced a folded strip of rubber tissue to the apex of the petrous pyramid. The fundus oculi had previously been normal. In twenty-four hours after the introduction of the folded rubber tissue, there were distinct evidences of a papilledema. This disappeared in twenty-four hours after the removal of the rubber tissue. If the very slightly increased intracranial pressure incident to the introduction of a rubber tissue drain could cause such rapid changes in the fundus oculi, it is no wonder that pathological conditions either of an inflammatory, circulatory, or a neoplastic nature will give rise to similar conditions.

The changes which take place in the fundus then are of a two-fold nature. First, those which are the direct result of increased intracranial pressure, that is, changes due to a mechanical cause, pure and simple, and also changes which are of an inflammatory nature, that is, those in which we have an actual neuritis of the optic nerve fibers.

There is no reason as far as I can see why these two conditions may not co-exist, that is, they may have a papilledema due to pressure and in addition to this have certain changes in the nerve itself due to inflammatory changes within the cranium, that is, a neuroretinitis. In a recent case of sinus thrombosis operated upon in which an extradural abscess was found at the time of operation the examining ophthalmologist pointed out quite clearly that in one eye we had a simple papilledema while upon the opposite side there were in addition to a papilledema certain evidences of a neuritis of the optic nerve. He even went so far as to suggest an accompanying meningitis, although the patient had no symptoms of meningitis. I explained to him that the only area of dura exposed was a small area over the lateral sinus, yet to his mind this was quite sufficient to have caused the inflammatory changes in the optic nerve itself.

The records of my own cases have been incomplete as far as this is concerned, and I cannot say whether in all cases of meningitis, inflammatory changes in the optic nerve are present or not. I do know, however, that in a great majority of these cases we have a papilledema whether or not the inflammatory changes are present. In the constitutional diseases mentioned above, lues, diabetes, nephritis and arteriosclerosis, the optic disc change is not limited to a papilledema. In fact, in many of these diseases this condition may be wanting, although other changes perfectly characteristic may occur in the optic papilla. As the discussion tonight is entirely on papilledema, the fundus changes which occur in the conditions just mentioned naturally do not come under discussion. They are mentioned, however, in order to emphasize the vast amount of information which may be obtained from an examination of the fundus oculi in each case.

Until I started to write this paper I do not think that I appreciated what a great advantage it would be to every otologist if he were to familiarize himself with the use of the ophthalmoscope and thus be able to inspect the optic papilla in each case of middle ear suppuration which came under his observation. I would even go further than this and say that it would be of value to examine the fundus in each case presenting aural symptoms which were not entirely due to a nonsuppurative middle ear condition. It is only recently that the true value of an extensive examination of the integrity of the eighth nerve in locating intracranial neoplasms has been fully appreciated. I for one feel that it would be very wise to be familiar enough with the use of the ophthalmoscope to give a fairly intelligent opinion on the condition of the fundus in every case.

40 West 51st Street.

PAPILLEDEMA IN RELATION TO OPHTHALMOLOGY.

DR. CONRAD BERENS, New York.

Owing to the fact that in this day of specialization the ophthalmologist is frequently the first physician consulted when a patient complains of headache and in practically all cases when the vision is disturbed, he is the first, it is extremely important that he should make an early diagnosis of papilledema, and thoroughly understand the management of this condition. It is less than seventy-five years since the ophthalmoscope was discovered by Helmholtz and our clinical knowledge of papilledema, by objective examination, has only been possible since its discovery. Ophthalmology following the discovery of the ophthalmoscope has acquired an extremely important relationship with other specialties in the diagnosis and management of papilledema. It hardly seems possible that less than 100 years ago Mackenzie,[1] who in 1833 wrote what was then considered one of the most authoritative books on diseases of the eye, should speak of blindness from inflammation of the brain, the consequence of suppressed perspiration. He also speaks of blindness from morbid formation of the brain. "The diseases referred to are tumors, formed by thickening of the membranes of the brain, or by the deposition of new matter between their laminae, or on their surfaces; also, tubercles, hydatids and fungous growths." He quotes Beer as saying, that the amaurosis resulting from morbid formations in the brain generally attacks both eyes at once, that the pupil is first contracted but later the pupils become dilated, and the vision is lost. He does not indicate how these tumors produce blindness, but believes that the physician should take a hopeful attitude in these cases, and that mercury and arsenic are particularly beneficial. Progress has been made in the treatment of papilledema since Mackenzie's time; but early diagnosis which is so important in treatment is sometimes difficult to make. Papilledema or choked disc is the term usually applied to a noninflammatory swelling of the optic nerve head, which may be as great as 3 m.m., which equals 6 or 8 dioptres of elevation when the apex of the edematous disc is compared with the fundus. Greater swelling is not seen, probably because the tissues of the nerve cannot be stretched more than this. It may be possible, as White[2] suggests, to restrict the term "papilledema" to

*Read before the Section on Otology, New York Academy of Medicine, Feb. 12, 1926. Symposium on Papilledema.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication May 13, 1926.

the edema of the disc caused by increased intracranial pressure, and designate those conditions due to foci of infection as "optic neuritis with edema," but in the early stages of these conditions we believe this is a difficult diagnosis to make, both clinically and pathologically. There are three principal theories to account for the presence of papilledema; the inflammatory, the chemical or toxic, and the mechanical theory. Experiments on dogs conducted in the Department of Experimental Surgery of Columbia University, with Dr. Grace, leads me to believe that this theory, combined with the toxic or inflammatory theory, offers the most satisfactory explanation. These experiments were undertaken at the suggestion of Dr. Tilney in the hope of determining what part, if any, the supraoptic canal, described by Tilney,[3] played in the pathogenesis of papilledema. The canal was discovered in studying the forebrain of the domestic cat, and was seen as a lateral projection from the cephalic extremity of the third ventricle upon the dorsum of the optic chiasm and optic nerve. It is lined with ependyma, and is 0.75 m.m. in length. The embryonic basis of the supraoptic canal is to be found in a small tubular extension from the third ventricle, which Tilney has seen in many forms other than *felis domesticus* and *homo*. Tilney has noted the greater constancy of the presence of papilledema in cases of internal hydrocephalus than in brain tumor, and believes that this may be due to the pressure of the supraoptic canal on the chiasm and the optic nerves. We attempted to produce internal hydrocephalus experimentally by trephining the skulls of dogs, and injecting aleuronale into the lateral ventricles. We hoped to produce inflammation resulting in chronic internal hydrocephalus by obstructing the aqueduct of Sylvius, the foramen of Monro or the foramen of Magendie. This we were unable to do in the three dogs injected, surviving after the operation. Thomas,[4] whose work we attempted to duplicate, was successful in the large majority of experiments and papilledema frequently accompanied the internal hydrocephalus. We were also unable to produce papilledema, although the central vein became dilated, as did the capillaries on the discs, through raising the intraventricular pressure to 150 m.m. of mercury by injecting salt solution eleven days after injecting aleuronal. However, by repeating the experiments of Cushing and Bordley[5] and raising the pressure in the subdural space by injecting salt solution we were able to produce edema of the discs, which could be partially reduced by pressure on the eyeball, even though we found no staining of the tissues of the eyes when methylene blue was used, in this manner, or when injected into the lateral ventricle. We believe, therefore, that the mechanical factor in the production

of papilledema is important, and probably acts by obstructing the vitreous outflow along the perivascular lymph spaces, although we realize the difficulty, even in tumors, of absolutely ruling out the presence of a low grade toxic inflammation, and believe that the altered cerebrospinal fluid may produce sufficient inflammation to block the fine lymphatic channels. In intraocular growths, for example, sarcoma of the choroid, we frequently see intraocular inflammation, which we believe to be due to the toxins from the tumor. Parker[6] also believes in the mechanical theory of the production of papilledema, for he has shown, by experiments on dogs and monkeys, that papilledema is greater in the eye with the lower tension, even though the experimental lesion was placed on the opposite side to the eye which had reduced tension. The intraocular pressure was lowered in these eyes by the trephine operation. Parker[7] has studied the tension of the eyeball in relation to twenty-three proven cases of brain tumor. In six cases the swelling was greater in the eye of lesser tension; in ten cases the swelling was equal in the two eyes, and the tension was equal in both eyes; in five cases there was the same amount of swelling in both eyes, but the tension in the eyes was different, although the appearance of the nerves was the same in both eyes. He classified this last group as an exception to the rule and states that of the cases examined 78.2 per cent conform to the rule, and 21.7 per cent are exceptions. As Parker's experimental observations gave 100 per cent of cases which conformed to the rule, and the greater amount of swelling appeared in the eye with the lesser tension in 78 per cent of the clinical cases, and as this point has been confirmed in these cases we have seen, it appears that no deduction can be made in regard to the site of the lesion from the degree of the swelling of the optic nerve. Further, we have no way of diagnosing from the papilledema either the site or extent of the lesion or the probable time which the lesion has existed. We have studied the intraocular tension in four of our patients with papilledema in whom the presence of a brain tumor was verified at operation, and in two cases the pressure was unequal, and the swelling of the discs was greater in the eye with the lower tension; in one case the amount of swelling was the same, and the tension was the same in each eye. The fourth case is reported in detail in this paper and the papilledema, which was unilateral, was noted in the eye with the higher tension. The differential diagnosis between papilledema and papillitis in the early stages is one which may well tax all the resources of the ophthalmologist and neurologist. We are familiar with the fact that the vision is apt to be diminished in the eye, with the papillitis out of proportion to the

degree of swelling, and that the vision in a patient with papilledema may be very little affected; further, that the degree of swelling in papillitis rarely exceeds 1 m.m. or 3 diopters. There are three other points that may be worthy of consideration. The first point is the question of the enlargement of the blind spot. The enlargement in papilledema is usually not as great as in papillitis in proportion to the degree of swelling of the nerve. This point may be easily understood when we consider that in the first instance the enlargement is frequently only a mechanical condition, due to the spreading aside of the rods and cones at the entrance of the optic nerve into the eyeball, while in the second instance the enlargement is due to the depressed function of the nerves themselves as the result of the action of some poison.

Study of the blind spot is extremely important in differentiating pseudopapillitis from papillitis and papilledema, for in pseudoneuritis the blind spot is extremely small. It is probable, as has been previously stated,[8] that the blind spot is examined most accurately on a dark gray screen, illumined by artificial daylight, but a simple but accurate method, provided the observer's blind spots are normal, is as follows:[9] The patient holds one end of a string 100 c.m. in length against the cheek below the right eye over the malar bone, while the observer holds the other end in the same position below his left eye. The eye not under examination is covered. A white-headed pin or point of light is tied into or passed through the center of the string. The test object is carried temporally slightly below the horizontal, eye fixing eye, until the blind area is found and compared with the observer's. This method is rapid but accurate, for it controls distance and fixation, and with a little practice will be found to be a helpful clinical test.

The second point is that in Biallart's[10] new pressure guage* we have a way of determining very early the presence of increased pressure in the vessels of the eye, and we believe that this should prove to be an extremely important point in the differential diagnosis of these conditions. If there is marked increase in the diastolic pressure in the retinal arteries as compared to the general diastolic pressure in the brachial artery, it is almost certain to mean that the pressure of the spinal fluid is raised. This is an extremely important point if Bordley[11] is correct in assuming that choked disc is always dependent upon an increase in intracranial pressure.

The normal formula as given by Magitot[10] is as follows: "It is the comparison between the figures of the local and general blood

*Baillat's pressure guage may be obtained from Pirard and Coeurdevache, Rue Blainville, No. 7, Paris.

pressure which alone has a clinical value, and the following rule which constitutes the normal formula should be remembered:

a. The normal arterial general pressure and local arterial pressure are to each other as 0.45 is to 1. Ex.: General diastolic pressure, 60 m.m. Hg. Local diastolic pressure, 35 m.m. Hg.

b. The high general arterial pressure and the local arterial pressure are to each other as 0.50 is to 1. Ex.: General diastolic pressure, 100 m.m. Hg. Local diastolic pressure, 50 m.m. Hg.

In choked disc the formula is as follows: The general blood pressure: Diastolic, normal. Systolic, normal.

The local blood pressure: Diastolic, high. Systolic, often normal.

The study of a certain number of clinical cases has moreover shown that every time this formula has been met with, there was a hypertension of the spinal fluid, even if there was no choked disc. It is therefore clinically possible to discover an important pathologic phenomenon before the appearance of the objective sign which is a consequence of it.

Here is an instance: A man who had been wounded in the war, and who showed some symptoms of Jackson's epilepsy, complained of pains in the head. There was no choked disc, but I found an ocular tension of 30 m.m. Hg., a general diastolic pressure of 70 m.m., Hg. and systolic of 135 m.m. Hg.

The local diastolic pressure was 70 m.m. Hg. The normal formula (as 0.50 is to 1), no longer existed since the two diastolic pressures, local and general, were identical.

We diagnosed great hypertension of the spinal fluid. Puncture in the lumbar region justified this diagnosis, and by means of radiography a small intracerebral projectile was discovered." Findings with Baillart's pressure guage should be checked against spinal fluid pressure as studied with the spinal mercurial manometer, whenever spinal puncture is not considered too dangerous.

The third point is one recently suggested by White[12] from his study of optic canal radiographs in relation to retrobulbar neuritis. "Neuritis, either acute or chronic, in the normal or abnormally large canals, seems to be usually of extranasal origin. This, if it proves true, should be of great importance in making a differential diagnosis between intracranial, multiple sclerosis, luetic and accessory sinus cases."

As soon as papilledema is discovered, the etiology should be determined, which frequently requires the aid of several consultants. The ophthalmologist should carefully note the corrected vision, blind spots and visual fields with standardized illumination, and small test objects and if he fears permanent loss of vision or visual field,

he should urge an immediately palliative decompression operation, even though the etiology is unknown. That syphilis has been ruled out is understood, for we believe with de Schweinitz that medical treatment in these cases should be given a thorough trial before resorting to surgery.

We cannot urge too strongly the repeated careful study of the visual fields, using small test objects and standardized conditions, for it has been our experience that visual field changes sometimes give the first localizing signs. We agree with Elsberg[13] that papilledema should be considered a late, rather than an early sign of brain tumor. He gives the following figures in regard to the presence of papilledema at the time of the first examination in cases which later were proven to have brain tumor by operation or at autopsy. The fundus was normal in 24; papilledema of 1 D., 6; papilledema of ½ D., 11; papilledema of ¾ D., 13; papilledema of ⅝ D., 9.

The differential etiological diagnosis of the cause of papilledema is extremely difficult in some cases, and the conditions which are believed to produce papilledema are the following:

1. *Brain Tumors* (including gummata, tuberculomata and brain abscesses), according to White[2] 58 per cent of brain abscesses arising from middle ear infections are accompanied by optic nerve changes.

2. *Tumor Equivalents.* According to Horrax[14] this term should be applied to conditions simulating brain tumor, for which an intracranial operation is indicated just as positively as in tumor.

a. Subacute or chronic cisternal arachnoiditis. (Serous or cystic meningitis.) Etiology unknown but possibly a cerebral infection allied to poliomyelitis. Differentiated from tumor only by an exploratory operation.

b. Oxycephaly.

c. Pachymeningitis hemorrhagica interna.

3. *Sinusitis.* Seventy-five patients with sinus disease and optic nerve involved were studied by White.[2] Three showed papilledema of low degree, 2 to 3 diopters, but Sluder speaks of 6 to 8 diopters of swelling in a patient with bilateral sphenoiditis.

4. Arteriosclerosis. (It is a question whether a toxin is present in these cases.)

5. Encephalitis.

6. Tuberculous meningitis.

7. Meningitis from other infections.

8. Intracranial aneurysm.

9. Cerebral thrombosis.

10. Nephritis. (Possibly the same circulating toxin which causes papilledema also causes nephritis.)

11. Lateral sinus infections. White[2] says 40 per cent show optic nerve changes.

12. Infections from teeth and tonsils.

13. Cerebral edema and hemorrhage following cranial trauma.

14. Lead poisoning was the cause of papilledema in 0.3 per cent of the cases studied by Uhthoff.[21]

That the differential diagnosis may be difficult even in a unilateral papilledema is well demonstrated by the following case seen by several consultants. Mrs. G. C. D., age 51 years, consulted us on Jan. 14, 1924, complaining of difficulty in reading and threading a needle, vision of right eye was 20/20, left eye 20/20, muscle balance and muscle strength were normal, and with a plus two sphere the patient read Jaeger No. 1 type at 300 m.m. in each eye. Muscle balance and muscle strength were normal; tension, fields, lids and ocular movements were normal. The corneal sensitivity was normal, the media were clear, but there was .5 D. swelling of the right disc, with blurring of the outlines of the disc. The pupils were equal and the irides reacted normally to light, accommodation and ciliospinal stimulation. There was no retrobulbar tenderness, the right blind spot was slightly enlarged, the field for color in the right eye was slightly contracted. The Wassermann test, the examination of the tonsils and nasal sinuses by Dr. Erskine, and the neurological examination by Dr. Casamajor were negative. Dr. Casamajor suggested spinal puncture, but the patient refused. The Roentgenograms of the teeth were as follows: inferior left first molar canals partly filled, peridental membrane about apex of mesial root thickened (periodontitis pus pocket involved half of roots). Roentgenogram of the skull by Dr. Law was reported as follows: the frontals are small, with thick anterior wall; they show a mild thickening of the membrane; the ethmoid capsule is well back, and shows some slight periostitis in the cell walls; the ethmoids show mild involvement at this time; the left antrum shows moderately severe involvement; the right antrum is clear; the sphenoids are large, extend well forward, and show mild thickening of the membrane. This is a mild chronic sinusitis. The sella is elongated and depressed with some absorption of the posterior clinoid. The outline is regular but there is a small calcareous deposit in the pituitary. . . . The evidence points to a pituitary tumor. Dr. Mersereau, who made the general examination, reported blood pressure 140/95, the heart slightly enlarged and early interstitial nephritis; the lungs

were normal, and the complement fixation test for tuberculosis was negative. We thought it best to have other Roentgenograms of the head and sinuses, and Dr. A. H. Busby reported. Examination of the head shows the sella definitely enlarged, the bowl of the sella deep and long, and the posterior horns are slender, and show erosion, and the distance between the anterior and posterior horns is increased. This is indicative of a tumefaction of the pituitary, that is, abnormal enlargement of the pituitary gland with a thinning of the sella wall and erosion of the posterior horns, the sphenoids and posterior ethmoids all appear slightly cloudy. Dr. C. W. Field reexamined the blood and urine and reported: this patient shows lowered renal functions of all substances tested, and figures in the blood that are above normal for everything except creatinin, the most marked abnormality is that for salt. The patient complained of severe headaches, which were throbbing in character, and as Dr. F. W. Janney, of Baltimore, had operated on her nasal sinuses several years before we examined her, he was asked to see her again. He reported that he could find nothing in her sinuses. Re-examination April 1, there was no change in the vision, but the blind spot in the right eye was larger and the swelling of the right disc fluctuated between 1 and 2 D. As the diagnosis was confused by the possibility of a pituitary tumor, chronic interstitial nephritis and infection of the nasal accessory sinuses, the patient was asked to consult Dr. Harvey Cushing. He reported that the diagnosis lay between a tumor and a possible chronic arachnoiditis and that although he did not like to perform lumbar puncture on people with choked discs, nevertheless he thought it was justifiable in her case. An immense amount of fluid, 60 c.c., was withdrawn without upsetting the patient, and he thought this pointed almost certainly to a chronic arachnoiditis, and advised decompression if the swelling did not subside. On April 23 he reported the spinal fluid finding negative, one cell, no globulin, negative Wassermann, gold curve negative; in short, nothing whatever suggestive of tumor. We did not feel justified in advising measures other than nasal treatment and the extraction of teeth, which had been done without apparent benefit to the condition of the eye. As sinus infection was still suspected, we asked the patient to go to a high dry climate for the summer, and she took a trained nurse, who made careful notes of her vision, visual fields and blind spots, and reported to us at weekly intervals. When the patient returned in September there was only 1 D. of swelling of the disc, and the blind spot was not quite so large. We saw the patient on Jan. 22, 1925, when she came complaining of throbbing headaches, which she had not had since the

early part of her rest in the mountains. We suggested the use of mixed catarrhal vaccines, to which the patient was extremely sensitive, but which apparently stopped the headaches, and made nasal breathing more easy. Since giving these injections the patient has not complained of headaches, and the blind spots have become practically equal in size. It is now two years since the first symptoms were noted, and the patient states that she has no headaches, and that she has never felt better in her life. We find the vision normal in both eyes, normal visual fields, slight enlargement of the right blind spot, and 0.5 D. of swelling of the right disc, with slight blurring of the margins. The tension with the McLean tonometer was 25 m.m. in the right, and 18 m.m. in the left eye, which does not conform with the rule that the swelling is greater in the eye with the lower intraocular pressure, but in this case the edema was unilateral and had existed two years before the tension was taken.

Summary: A patient with unilateral papilledema, chronic interstitial nephritis, apical dental infections, possible sinus infection, probable pituitary tumor as shown by two Roentgenograms, and signs pointing to brain tumor, or arachnoiditis. The papilledema persisted for two years, and was apparently unimproved by the extraction of teeth with apical infections, nasal treatment, by spinal puncture, or by drugs. Mixed catarrhal vaccines seemed to be beneficial, whether by their nonspecific protein effect, or by their specific effect on bacteria in the nasal sinuses, or on some other focus of infection which was undiscovered, is not known. Possibly the injections of vaccine had nothing to do with the improvement, and the patient has a brain tumor which will be found to cause trouble later. This case points to the possibility that decompression operations could be done too early, and that patience with careful observation might show that in some cases operation is unnecessary, for after two years this patient has 20/15 vision in each eye, and normal visual fields. We believe, however, that the ophthalmologist must frequently be the one to decide when to perform decompression, and should base his decision on close observation of the vision and visual fields.

Treatment of Papilledema: This should be directed to the removal of the cause if this can be determined. From our experience, the great majority of papilledemas are due to brain tumors, or "tumor equivalents,"[14] which is particularly true of bilateral cases.

We believe the most important point in the treatment of papilledema from the standpoint of the ophthalmologist is the preservation of vision, and it is possible that resection of the optic nerve

sheath as suggested by Muller[15] may be considered in some cases where palliative decompression is indicated, for he claims relief of the symptoms due to cerebral pressure as well as amelioration of the papilledema.

Although we have stressed the fact that it is possible to perform a decompression too early, it has been our experience that the operation has more frequently been performed too late to save useful vision. As Marcus Gunn pointed out long ago, the visual prognosis is good if treatment is successful in the first three stages of choked disc, that is before massive exudates and hemorrhages appear in the retina, but when the edema begins to subside, and atrophy of the nerve is noted, the visual prognosis is bad. Sometimes valuable time is lost in attempting to make an etiological diagnosis, and Cushing's[16] statement that over 4 diopters of elevation must be caused by mechanical pressure, is important to remember in this connection.

Another point in regard to treatment is one raised by White[12] in regard to optic neuritis. He believes that small optic canals as shown by radiograms predispose to optic atrophy and that in normal or abnormally large canals recovery without atrophy usually results. Walker[17] has recently accounted for the presence of unilateral optic atrophy after encephalitis by the fact that the canal was smaller on the side of the atrophy. With this in mind we may find it necessary to operate on our patients who have small optic canals early, and for this reason the size and shape of the normal optic canal should be determined in each case, and compared with the normal findings.[18] In treating cases with undetermined etiology, it may be permissible to use some substance for its nonspecific effect in addition to the time-honored remedies like iodin, arsenic and mercury. Heine[19] has used tuberculin routinely, and we use autogenous vaccines made from the nose, sputum, tonsils, teeth and intestinal tract, or chemically killed stock, mixed catarrhal vaccine, particularly in cases where chronic sinusitis is suspected. We try to keep the dose below the point of producing anything but the slightest local reaction, and in extremely hypersensitive patients we first try to desensitize them nonspecifically by extremely small doses of some other nonspecific substance. In using autogenous vaccines, we have sometimes given vaccine containing only bacteria to which the patient was shown to be hypersensitive by intradermal tests as suggested by Thomas.[20] We are not certain that autogenous vaccines, even though selected in this manner have given better results than stock vaccines, and it makes us believe that many of our so-called specific treatments with vaccines are really nonspecific in action.

CONCLUSIONS.

1. Early diagnosis of papilledema is important and in the incipient stages it may be difficult to differentiate from papillitis and pseudopapillitis. Papilledema should make one strongly suspect the presence of brain tumor, but it should be considered as a late, rather than an early diagnostic sign.

2. Greater swelling of the disc is usually seen in the eye with the lower tension, but exceptions frequently occur.

3. No important diagnostic conclusion can be made from the degree of swelling of the disc as to the probable location of a cerebral lesion, its extent, or the length of time it has existed.

4. The mechanical theory of Manz and Schmidt-Rimpler combined with a toxic factor seems to offer the most logical explanation of the production of papilledema. Edema of the disc may be produced by quickly raising the subdural pressure and increasing the amount of fluid at the same time. This probably acts by blocking the outflow from the vitreous by compressing the perivascular lymphatic spaces, as we have failed to stain the intraocular tissues by subdural, subarachnoid, or lateral ventricle injections. Clinical evidence of the influence of pressure is furnished by the rapid subsidence of the edema, in most instances, following decompression and the fact that the swelling of the disc is usually greater in the eye with lower tension. Tilney's theory of the influence of the supraoptic canal in the production of pressure on the chiasm and optic nerves may be important in the production of edema in chronic internal hydrocephalus, but quickly raising the intraventricular pressure to 150 m.m. of mercury only produced congestion of the intraocular veins. We believe that a toxic or inflammatory factor is present in most, if not all, cases, and probably acts through the altered cerebrospinal fluid by producing a low grade inflammation of the perivascular lymphatic channels at their exit from the eyeball.

5. Early operation may be indicated in patients who have small optic canals as shown by Roentgenograms.

6. In treating papilledema neither the etiology nor the question of which theory in regard to the mechanism is correct, is as important to the ophthalmologist as the question of saving useful vision. Therefore, if loss of vision is feared in papilledema and simple methods of treatment have failed, decompression should be performed if the pressure of the cerebrospinal fluid is raised. The procedure is practically without danger, and may save useful vision.

7. In papilledema of undetermined etiology, it may be justifiable to inject vaccines or some other protein for the nonspecific effect in

raising cell resistance, in addition to using arsenic, mercury and iodin, and performing spinal puncture.

8. Surgical intervention in regard to the question of saving useful vision is more frequently resorted to, too late, rather than too early, in the treatment of papilledema.

9. In Baillart's new pressure guage we have a way of determining the presence of increased intracranial pressure at an early stage. If these findings are confirmed by the spinal mercurial manometer, when spinal puncture is not considered too dangerous, early treatment of the causes of papilledema may be possible, for it is our opinion that choked disc is usually a symptom of high intracranial pressure.

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30 East 40 Street.

PAPILLEDEMA FROM THE VIEWPOINT OF THE RHINOLOGIST.*

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The association of paranasal sinus disease with retrobulbar neuritis is by no means a recent observation though the subject has provoked a great deal of discussion in recent years. In 1886 Berger and Tyrman reviewed twenty-six cases of blindness due to sphenoidal disease. Fliess in 1895 reported curing a case of retrobulbar neuritis by operating on the sphenoidal sinus. Cases of optic atrophy were also reported associated with sinus disease where the earlier inflammatory stage had not been observed. For several years after this period an occasional case would be reported, but the general recognition by oculists and rhinologists that sinus disease and retrobulbar neuritis existed so often in the relation of cause and effect did not occur till the last decade. Even now there is some suspicion that this relation is neither recognized nor appreciated as fully as it should be even among specialists, especially in regions remote from large medical centers, and very few of the general practitioners ever seem to have heard of it.

That sinus disease can cause a retrobulbar neuritis and that the condition can be cured by opening the posterior sinuses is surely a well established fact and no one who has had much experience can possibly doubt it.

By his writings Leon White, of Boston, has not only added a good deal to our knowledge of these associated phenomena, but has done a great deal to establish the present orthodox standards of investigation and treatment.

Let us now consider the anatomical relations of the optic nerve to the posterior sinuses. The nerve enclosed in its bony canal passes through the upper and outer part of the external wall of the sphenoid or through the external wall of a posterior ethmoid cell. It may present the entire circumference of the canal within the cavity or as is more usual, a portion only is within the sinus. It may pass through both sphenoid and posterior ethmoid. The thickness of the bony covering of the nerve varies greatly, in some cases being entirely absent. Neivert in a series of 220 dissections found dehiscences in 12. It can thus be seen how easily an

*Read before the Section on Otology, New York Academy of Medicine, Feb. 12, 1926. Symposium on Papilledema.

Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication May 13, 1926.

inflammatory process in these cavities can extend to the optic nerve. White has called attention to the size of the optic canal as a factor in producing symptoms and has postulated this finding as a valuable indication in the treatment, considering that the finding of an optic canal with a 4 m.m. diameter or less demands more immediate operation than a larger canal, and that one as large as 5 m.m. can be more safely considered amenable to treatment and that these cases will be far less likely to go on to optic atrophy. I have had no experience with this method of examination, but I frankly confess that I am always skeptical of any method which reduces diagnosis and prognosis in human disease to the exactness of mathematical formulae.

When we come to consider the pathology of the sinuses in these cases we seem to find a notable absence of those lesions which we would expect to find. Caries and necrosis with polypi which would seem to be the most likely lesions have never been present in my cases, nor have I seen the report of any such cases. I have seen a purulent discharge present in only three cases and of these two recovered completely on treatment. It is generally considered that the hyperplastic form of sinusitis is the one most commonly associated with involvement of the nerve. I have found this type of nondescript pathology in several cases. I can recall three cases with granulation tissue filling up the cells, giving them a doughy consistency with little feeling of cell structure during the operation. One case showed several tiny polypi within the ethmoid cells where no suspicion of polypi was present in the nose. I have opened three cases where sinuses were suspected by the oculist and found no appearance of any abnormality. There was no improvement in the eye condition in these cases. One of them had her tonsils out a week later and began to improve slightly. One finding which has been fairly constant in my cases, and others have reported the same thing, is a bad anatomical condition in the posterior part of the nose, a septum crowding over the posterior end of the middle turbinate thus interfering with drainage from this region. I had noticed this in the majority of cases before knowing that Stark and White had noted the same thing.

The diagnosis of sinus involvement in these cases is very obscure. The history is important. A nasal infection anywhere from a week to three months previous to the onset of eye symptoms has been fairly constant in my experience, but often no evidence of it is found on the nasal examination. The anatomical condition above referred to and especially with a history of recent infection has

been the usual foundation for suspecting the sinuses as the probable cause, provided no other foci throughout the body have been found and a thorough inquiry made in the history as to the possibility of any chemical poisoning, such as lead, arsenic or alcohol. The X-ray has been a great help. There have been in several cases positive findings of thickened tissue in the ethmoids with loss of definition of intracellular septa, and this has been verified by operation. In two cases I can recall the nasal examination was negative and the X-ray showed evidence of a pathological process. It is well to remember that it is not only the proximal sinuses which can produce papilledema. An infected antrum may cause the same phenomenon. I have had one case where washing out an obviously infected antrum brought about a quick recovery of the nerve. The patient had ethmoid involvement as well but still has it, and I am inclined to think the nerve condition was solely due to the absorption from the antrum. We know also that infection from the tonsils and teeth can both produce the same changes in the nerve.

The way these various lesions produce the secondary nerve lesion is a much discussed subject. In the case of the sinuses one might with some reason think of the possibility of a pressure effect, especially in a case where there might be a dehiscence in the bony optic canal. The rapidity of cure in some cases would lend credence to this view. In all cases that I have seen I could not determine on operation the slightest evidence of pressure either from fluid or a greatly swollen or degenerated membrane. As you know, the mucous membrane of a sphenoid can swell up to fill the whole cavity. I have seen this subsequent to an operation, but never have noticed it at the time of operation. I am inclined to think that it is either a direct extension of inflammation, as may occur from the sinuses, or a toxic phenomenon due to absorption. The same quick recovery may follow the extraction of a tooth, as will follow opening sinuses. In the two cases I have seen where the tonsils were the probable cause, recovery was not so immediate. One can argue that if it is ever due to absorption, why is it not a usual bilateral phenomenon? That is not necessarily the case as metastatic foci elsewhere show no system or symmetry in their appointed localizations. Besides, papilledema may be a bilateral phenomenon and I recall three such cases. Involvement was not equal on both sides and one antedated the other. The investigations of recent years have tended to regard phenomena due to so-called absorption as a true metastasis with the actual migration of the organisms from the primary to the secondary focus. This happens, as we know, in syphilis and it may be taken as the typical focal infection, though it leaves very little

trace of its primary invasion, and no doubt some of the others act in the same way; hence the difficulty in finding their original field of invasion.

We must now pass on to consider the treatment of these cases and this is a matter often requiring promptness of decision and wise judgment. Granted that the diagnosis has been made by a competent oculist and everything eliminated as a cause outside of the field of laryngology. He is confronted with the task of balancing the probability of diseased tonsils or sinuses as a cause. If the oculist can tell you that the nasal half of the nerve is the part most involved it will be considerable evidence in favor of the sinuses. If this is not apparent one will only be guided by which region shows the most pronounced evidence of disease. There is usually a necessity for prompt decision and I would not hesitate to advise that in a given severe lesion with complete loss of vision where both tonsils and sinuses are suspicious, the tonsils should be removed and the posterior sinuses opened at the same time on the side affected. If on the other hand the lesion has been found early and vision has not depreciated so greatly and there is very obvious disease of one part to the probable exclusion of the other, the most diseased part should be operated on first and if no improvement occurs in a week the other region should be attacked. In cases of some duration, where hope of recovery is very remote, the elimination of all possible foci should be effected as quickly as possible.

I have so far been considering operative treatment but there is a class of case in which treatment may be tried first, young subjects with an early lesion. I have had three such cases which recovered on intranasal treatment, *viz.*, shrinking up the nose, hot saline irrigations and argyrol, 10 per cent. This treatment should not be continued more than a week unless the process in the nerve is beginning to show signs of recovery. A diseased antrum should be washed out before attacking the other sinuses and if any improvement occurs this may be repeated and may be all that is necessary. In operating on the sinuses it is always advisable to do a submucous first and I usually do a complete exenteration of the ethmoids on the side affected, making a large opening in the anterior sphenoid wall.

Prognosis depends on the amount of damage done to the nerve and the recuperative power of the individual. As a general rule the earlier the lesion and the younger the subject the better the prognosis, but never despair of any case as there may be some slight improvement in the apparently hopeless. I saw some recovery in a

case once by opening the sinuses where vision had been gone for a month. I also saw slight improvement from removing the tonsils where vision had been practically gone for three weeks.

I wish to report briefly a case of papilledema which came to me last June with the following history: A girl, age 13 years, had an attack of what was called "flu" in February and had considerable nasal discharge at the time, which seemed to clear up satisfactorily. On April 4 an oculist was consulted on account of failing vision. He found a retrobulbar neuritis on both sides, with vision 20/40 in each eye. The condition in the left eye continued to get worse and the right eye remained nearly the same for the next two months.

When I saw her June 12 she had been advised to see Dr. Cushing with a diagnosis of suspected brain tumor. I examined her nose and found practically no evidence of sinus disease, but referred her to Dr. E. S. Thomson for an eye examination. He reported vision in right eye 20/40, left eye 20/200, with a marked retrobulbar neuritis on both sides, the nasal side of the nerve being most affected, and with contraction of the visual fields. I had an X-ray taken by Dr. Law and it showed marked thickening of ethmoids and sphenoid on both sides. On June 16, under general anesthesia, I did a submucous and opened ethmoids and sphenoids on both sides. Next day the right eye had 20/15 vision and in three days the left eye had 20/15. Both eyes have been normal ever since.

The duration of the lesion with so rapid a recovery was rather remarkable. The X-ray was a great help in the diagnosis and the findings at the operation corresponded. The ethmoids on both sides were thick and doughy, with no feeling of intracellular septa in the posterior regions.

101 East 58th Street.

NEURO-LABYRINTHITIS OF FOCAL INFECTION ORIGIN IN A CASE WITH A PLUS-FOUR WASSERMANN.*

DR. GEORGE W. MACKENZIE, Philadelphia.

The clinical picture of neurolabyrinthitis of focal infection origin and that of syphilis resemble each other sufficiently to warrant the closest kind of study to differentiate them. In looking for the etiologic factor in neurolabyrinthitis we cannot accept as conclusive the finding of a positive or a negative Wassermann alone; for cases of neurolabyrinthitis syphilitica exist, especially of the latent form, with a negative Wassermann. On the other hand, neurolabyrinthitis from a focal infection can exist in the presence of a plus-four Wassermann without the syphilitic virus contributing any appreciable influence; witness the case to be cited. The best proof of precision in the consideration of cause and effect is the promptness and permanency of the improvement or cure† of a disease that follows the removal of the causative factor in question. In no case is this more true than in neurolabyrinthitis.

Concerning the subject of neurolabyrinthitis, the writer has already written quite freely; notwithstanding, there still remains much to be said. The most gratifying results thus far obtained have been in cases of neurolabyrinthitis of focal infection origin referred, quite frequently, by other otologists because of vertigo and nystagmus that prompted the suspicion of some more serious intracranial condition. Such cases are by no means rare. The one to be reported is of that kind.

The case, S. W., male, age 52 years, physician, was first seen by the writer Feb. 20, 1926.

History: Between the age of 16 and 17 years the patient went through an attack of pneumonia with empyema. In the year 1918 he had an attack of "flu." He has suffered no other illnesses except-

*Read before the Michigan State Medical Society, Lansing, Mich., Sept. 14, 1926.

†The word "cure" cannot be made to apply to all cases. The reason is evident when we consider that long-lasting toxemias lead to chronic plastic inflammation with its concomitant metaplasia of the higher developed sense epithelium. Therefore, the more tardy we are in removing the source of the toxemia, the more fixed the pathologic changes have become; when the results take on more the form of "improvement" than of complete "cure." The complete cures occur in those cases only where the toxemia has not produced changes beyond those of congestion or mildly acute serous inflammation.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Oct. 4, 1926.

ing slight colds. About six weeks ago he had laryngitis, with infection in the nose and throat, which cleared up under treatment in a few days. His present illness began about three weeks ago with dizziness. He did not think it very important at the time and continued working very hard, as he had been doing for two weeks before. The dizziness not only continued but became more severe. After four days he began to ease up in his work. One evening there developed severe headache which lasted for about a half hour. His partner tested the blood pressure and found it 100 cystolic. The diastolic pressure was not recalled. The patient and his partner decided that the illness was due to overwork. However, he continued to work, but not so hard, for about three more days. The dizziness increased, but was less evident when he was quiet and sitting still. After a few more days he found that moving the head to the left side increased the dizziness very much. He then called in an ophthalmologist, who discovered that the eyes were normal with the exception of a "rotary nystagmus to the right." The optic discs were observed to be normal as well as the blood vessels in the fundi. His physician examined him later and "found intense congestion of the mucosa of the nose and the Eustachian tube blocked on the left side." His diagnosis was "acute labyrinthitis." Following this the patient claims to have had a specialist in internal medicine and a neurologist to examine him. At that time it was claimed the Romberg was normal. There was slight inco-ordination in the muscles of the legs. No headache and no pain of any kind since the beginning of his present illness except that already noted; diagnosis of acute labyrinthitis. The treatment of the nose was continued, and after a week the left Eustachian tube was catheterized. The feeling in the ear today, Feb. 20, 1926, is the same as it has been for the last two weeks. The ear feels as though it was "blocked." The hearing has been variable, at times "less than 50 per cent, and at other times considerably better." It has not been normal at any time. His voice sounds to him as though it was far away. The nystagmus has gradually improved. On Monday last, five days ago, another neurologist made an examination and diagnosed a "thrombus in the cerebellum," and spoke of having seen "one case in Boston similar to his with a negative Wassermann." The Wassermann was then taken and found to be plus-four. Before this the unanimous opinion was that of acute labyrinthitis, due probably to infection of the nose and throat brought on by overwork. It was suggested that a few weeks' vacation in Florida would fix him up, and transportation to and accommodations in Florida were arranged. Antisyphilitic treatment was started on Wednesday following the examination by the Boston neurologist.

About this same time it was decided to leave the whole matter, including the labyrinthine tests, in the hands of the writer.*

At the beginning of his present illness his head was X-rayed, but nothing abnormal was found. Last May, nine months ago, he was sick for two weeks with what was pronounced cholecystitis, but which the patient now believes was some syphilitic manifestation. For the past two years he has been a "teetotaler." Before that time he went through a two months' siege of drinking, the blame for which he put onto some heavy financial losses. Prior to that time he was but a very moderate drinker.

The teeth had been X-rayed often (every six months). The last time was at the beginning of his present illness. He is aware of the presence of several "dead" teeth, but denies having any pyorrhea. The dentist advised him to keep the teeth for a while. Temperature taken on this first visit was 99.2° F.

The functional hearing test findings elicited at this first visit are as follows:

A.D.		A.S.
Short 5"	← Weber	
Short 7"	Schwabach	Short 10"
+ 40"	Rinne	+ 45"
Short 13"	Air	Short 18"
Short 40"	C_1	Short 60"
Short 5"	c_4	Short 8"

Otoscopic examination of the right ear reveals a slight amount of cerumen, requiring removal before a satisfactory inspection of the deeper structures could be made. The drum membrane is intact, brilliant, of normal translucency, allowing the long process of the incus to be seen. There is no pathologic retraction; the mobility of the membrane is normal with the Siegel instrument. With Politzer inflation the membrane comes out over a large area without overlapping the stria malleolaris, and returns promptly to its primary position.

Otoscopic examination of the left ear presents no cerumen or other obstruction in the canal to interfere with the observation of the drum membrane. It is intact, brilliant and normally translucent, allowing the long process of the incus to be seen. The mobility of the membrane is normal with the Siegel instrument. With Politzer inflation

*The very complete history up to this point is the result of careful observation on the part of the patient. He was asked to take his time and write out the history fully. It was copied almost verbatim. The remaining history was elicited by questioning the patient.

the membrane comes out over a large area without overlapping the stria, and returns promptly to its primary position.

Examination of the nose shows the mucous membrane to be quite red on both sides. There is a pronounced deflection of the nasal septum to the right side, with a large spine along the suture line on the same side. After shrinking the mucous membrane with cocain, the left middle turbinate becomes visible. There is a free space between the left middle turbinate and the lateral wall and between it and the septum. The right middle turbinate also becomes visible after shrinking. It appears to be undersized because of the septal deflection to that side; likewise, the right inferior turbinate is undersized. There is a compensatory hypertrophy of the left inferior turbinate, together with a slight hyperplasia of the mucous membrane along its inferior margin and posterior end. There is no gross evidence of accessory sinus disease by anterior rhinoscopic examination.

Examination of the throat reveals a small papilloma at the base of the uvula on the left side. Both tonsils are submerged, and from the crypts cheesy deposits can be expressed with the pillar retractor. There is a slight secondary catarrhal pharyngitis present.

Examination of the teeth shows very evident pyorrhetic resorption. The upper right first molar is carious, and there are several other very questionable teeth which warrant a careful X-ray examination.

Examination for spontaneous nystagmus: When the patient looks to the extreme right side there is a fairly pronounced nystagmus to the right. When he looks to the extreme left side there is a fairly pronounced nystagmus to the left. However, there is this difference: The nystagmus to the right side is pre-eminently horizontal in character; whereas the nystagmus to the left side is more rotary, with the same angle deviation of the eyes. Upon looking straight ahead the presence of nystagmus to the right side is doubtful.

Galvanic test:

Right Ear	Left Ear
Kathode 4½ Ma. ↺	Kathode 9 Ma. ↻
Anode 10 Ma. ↻	Anode 10 Ma. ↺

Turning test, with the head erect: After ten turns to the left side there follows a horizontal nystagmus to the right, lasting for 12 seconds. After ten turns to the right side there follows a horizontal nystagmus to the left, lasting for 9 seconds.

With the head inclined 90° forward: After ten turns to the left side there follows a rotary nystagmus to the right, lasting for 11 seconds. After ten turns to the right side there follows a rotary nystagmus to the left, lasting for 5 seconds.

The patient was then sent to Dr. B. M. Wagenseller for a differential blood count and urinalysis, and to Dr. J. W. Post for an X-ray examination of the teeth. He was put on mercurial inunctions, and told to report in two days.

The blood findings, under date of Feb. 20, 1926, were as follows: Hemaglobin, 90 per cent; red blood cells, 5,180,000; white blood cells, 12,000; polynuclear, 53 per cent; small lymphocytes, 39 per cent; large lymphocytes, 5 per cent; eosinophiles, 3 per cent.

Urinalysis report by Dr. Wagenseller, Feb. 22, 1926, reads: Color yellow; reaction acid; spec. grav. 1031; albumin negative; sugar negative; indican normal; epithelia present; oxalates present; urates present.

The X-ray examination of the teeth revealed the following findings: The upper left third molar, upper left first molar, upper left second bicuspid, upper right second bicuspid and the upper right second molar are all pulpless teeth, showing evidence of chronic periapical disease. The lower right remaining molar is a pulpless tooth showing no evidence of canal filling in the distal root. This tooth should be looked on with marked suspicion, in view of all the other changes. The distal aspect of the root of the tooth is denuded of the alveolar process for about two-thirds of its length. There is a large area of caries beneath the filling at the neck of the tooth. There is also an area of caries on the mesial aspect. The entire alveolar process shows marked pyorrhetic changes and especially about the lower central and lateral incisor region.

Feb. 22, 1926: The patient reports for the second visit. He thinks his hearing has grown worse since the last visit. He also complains of more noises on both sides, but worse on the left. He complains, too, of a "stuffed up feeling" in the left ear.

The functional hearing tests were repeated on this visit, first by the writer and then by Dr. A. V. Mackenzie. The average findings for the tests are as follows:

Right Ear		Left Ear
Short 13"	Weber	
Short 7"	Schwabach	Short 18"
+ 39"	Rinné	+ 18"
Short 8"	Air	Short 40"
Short 40"	C_1	Short 90"
[illegible]	C_4	Short 8"

Feb. 25, 1926: *Examination for spontaneous nystagmus:* Horizontal nystagmus to the right when the patient looks to the extreme

right. Mixed rotary, horizontal nystagmus to the left when he looks to the extreme left. The nystagmus to the right appears to be a shade more pronounced than that to the left. There is a doubtful nystagmus to the right when the patient looks straight ahead.

Galvanic test repeated at this visit gave the following results:

Right Ear		Left Ear		
Kathode	5 Ma. ↺	Kathode	9 Ma.	No reaction
Anode	9 Ma. ↻	Anode	9 Ma.	No reaction

He complains of his mouth being quite sore, due no doubt to the combined influence of extractions and intensive mercurial treatment.

The report on the bacteriological examination made by Dr. Wagenseller under date of Feb. 27, 1926, reads as follows: Four teeth were cultured—upper right and left bicuspids; upper right and left molars. Growth was obtained from three, upper right molar giving nothing. Growth in all tubes was the same, as follows: Staphylococcus albus predominates; gram positive bacilli present; gram negative bacilli present.

On March 1, 1926, the patient reports that his hearing is slightly better, but is still "thick." The mouth feels quite sore, for which he was referred to Dr. Lloyd Strohm (Syphilologist), of Philadelphia.

March 2, 1926: Dr. Strohm reported that he had doscontinued the mercurial inunctions and instituted treatment with bismuth injections. From this time on, Dr. Strohm took charge of the treatment of this feature of the case. The patient reports that the vertigo is a little better today. He says that he can "walk better today than any day yet." The stopped up feeling in the left ear still continues. The lower right molar which the patient looked upon with suspicions has not yet been extracted.

March 12, 1926: The patient reports that after each extraction there is a temporary aggravation of his vertigo. The patient was so emphatic about this statement that it appears underscored in the original records. Owing to the extensive pyorrhea, in addition to the other pathologic changes noted by Dr. Post, it was deemed best to make a thorough clean-up of both upper and lower alveolar processes. Furthermore, it was considered advisable not to remove more than three teeth at one sitting, and then watch carefully for excessive reaction for a day or two following, before proceeding with further extractions. The worst appearing offenders were left for the last. The order of the extractions and after-treatment, taken from the records of the Exodontist, Dr. Cos Leefmans, is as follows:

Feb. 23, 1926: Extraction of three teeth; upper left first bicuspid, upper left first molar and upper left third molar. Apical abscesses were curetted thoroughly and cultures taken.

Feb. 24, 1926: Two teeth were extracted; upper right first bicuspid and upper right second molar ;thoroughly curetted sockets.

From Feb. 25, 1926, until March 4, 1926, the sockets of the extracted teeth were syringed daily. On the latter date one tooth, the lower right third molar, was extracted and pyorrhetic abscess drained and packed with gauze.

March 5, 1926: Treatment of gingiva with chlorodone.

Until March 10, 1926: Conservative treatment; besides three extractions with thorough curettment—the lower left lateral incisor, canine and first bicuspid.

March 12, 1926: Three extractions, with thorough curettment—the lower right cuspid and first and second bicuspids.

March 17, 1926: Two extractions, with curettment—lower left second bicuspid and first molar.

March 18, 1926: Four extractions* were made, with thorough curettment. The differential blood count was made one hour before, again on the following day (March 19), and again on March 20, reports of which appear elsewhere.

March 26, 1926: Extraction of two supernumerary teeth. After this date there followed daily treatment of the sockets.

April 9, 1926: Removal of sharp corner of process from the region of the upper left cuspid.

The blood count made just *prior* to the four extractions of March 18 netted the following findings: Hemaglobin, 98 per cent; red blood cells, 4,680,000; white blood cells, 13,200; polynuclear, 54 per cent; small lymphocytes, 35 per cent; large lymphocytes, 6 per cent; transitional, 1 per cent; eosinophiles, 4 per cent.

The blood count made the *day after* was as follows: Hemaglobin, 86 per cent; red blood cells, 4,600,000; white blood cells, 10,400; polynuclear, 55 per cent; small lymphocytes, 29 per cent; large lymphocytes, 7 per cent; transitional, 1 per cent; eosinophiles, 5 per cent; basophiles, 3 per cent.

*On this particular day our rule of "not extracting more than three teeth" at one sitting was broken, the writer having been present and cooperating. It was these particular teeth and sockets that the writer felt were most responsible for the patient's ailment and which required the most careful study, surgically and bacteriologically, and controlled by three different blood counts.

Two days after (March 20, 1926) the findings were: Hemaglobin, 90 per cent; red blood cells, 4,440,000; white blood cells, 8,000; polynuclear cells, 55 per cent; small lymphocytes, 23 per cent; large lymphocytes, 15 per cent; transitional, 2 per cent; eosinophiles, 5 per cent.

March 20, 1926: The patient reported that he felt generally much better. There was a very questionable spontaneous nystagmus to the right side, with no vertigo. He was still under treatment with bismuth, and also under the care of Dr. Leefmans for the after-treatment of his alveolar processes following the extractions, until April 9, 1926, when he went home, to report again in a few weeks.

On April 8, 1926, the blood Wassermann test made by Dr. Wagenseller was found to be plus-four as on former occasions.

April 9, 1926, the urinalysis was: Color, light yellow; reaction, acid; sp. grav., 1,025; albumen, faint trace; sugar, none; indican, marked; aceton, none; diacetic acid, none; epithelia, few; hyaline casts, considerable; granular, numerous; oxalates and urates present.

Under date of April 17, 1926, the patient sent a report of his condition, abstracts from which read as follows: "I am feeling very much better. The vertigo has continually improved. I might say that taking it week by week there has been a decided improvement in the vertigo each week for the past three weeks. An examination of the urine made April 14 showed no albumen, no hyaline casts, a few granular casts (3 or 4 in the whole field) and some cellular debris. An examination made today shows no change, with the exception that there are a few more granular casts."

Under date of May 6, 1926, another letter was received from the patient, abstracts from which read as follows: "I have followed the regulations carefully" (this referred particularly to the treatment of his syphilitic condition, which was now under the supervision of Dr. John B. Ludy, of Philadelphia, who was substituting for Dr. Strohm, who had gone to Europe), "am feeling much better and have gained three pounds since leaving Philadelphia, now weighing 184 pounds, which is fourteen pounds heavier than I weighed all last year. Urinalysis made yesterday showed practically no casts, no albumen and no debris."

June 9, 1926: The patient reports in Philadelphia that he is weighing more than he ever did, 186 pounds. He feels no vertigo, except rarely upon turning in bed from left to right. He feels slightly fatigued if he works too hard. He believes his hearing is good. The mouth has all healed up. Urinalysis is negative to albumen, casts and other pathologic findings. The functional hearing tests at this time are:

Right Ear		Left Ear
←	Weber	
Short 5"	Schwabach	Short 7"
+ 45"	Rinné	+ 47"
Normal	Air	Normal to questionably shortened
Normal	C_1	Normal
Normal	C.	Normal

Spontaneous nystagmus: Mixed rotary horizontal nystagmus to the right when looking to the extreme right. Mixed rotary horizontal nystagmus to the left when looking to the extreme left (physiologic). No nystagmus when looking straight ahead.

Galvanic test:

Right Ear		Left Ear	
Kathode	6 Ma. ↻	Kathode	9 Ma. No reaction
Anode	7 Ma. ↺	Anode	7 Ma. ↻ (?)

Turning test, with head erect: After ten turns to left there follows a horizontal nystagmus to the right lasting for 10 seconds.

After ten turns to the right there follows a horizontal nystagmus to the left lasting for 6 seconds.

Turning test, with head inclined forward 90°: After ten turns to the left there follows a rotary nystagmus to the right lasting for 9 seconds.

After ten turns to the right there follows a rotary nystagmus to the left lasting for 6 seconds.

Caloric test: After syringing the left ear with 2,000 c.c. of water at 60° F. over a period of 5 minutes, there follows no reaction in the form of nystagmus or otherwise with the head in an upright position or inclined in any direction.

Romberg is negative. The gait forward and backward, with eyes closed, is not as good as that of the average normal individual; in other words, it is more uncertain, and the tendency to stagger is greater in his case.

Sept. 2, 1926: The patient reports that he feels better than he has for years. He hears well and has no vertigo. He weighs 187 pounds, and is thinking of taking exercises to reduce. The blood Wassermann is still plus-four in spite of treatment.

Functional hearing tests:

Right Ear		Left Ear
←	Weber (?)	
Normal	Schwabach	Normal
+40	Rinné	+40
Normal	Air	Normal
Normal	C_1	Normal
Normal	C_4	Normal

Spontaneous nystagmus: When looking to the extreme right there is nystagmus to the right. When looking to the extreme left there is nystagmus to the left, but to neither side more pronounced than the other; hence physiologic. There is no nystagmus when he looks straight ahead.

Galvanic test:

Right Ear	Left Ear
Kathode 6 Ma. ↶	Kathode 7 Ma. ↷
Anode 7 Ma. ↷	Anode 6 Ma. ↶

Turning test: After ten turns to the left, with head erect, there follows a horizontal nystagmus to the right, lasting for 12 seconds.

After ten turns to the right, with head erect, there follows a horizontal nystagmus to the left, lasting for 11 seconds.

With the head inclined forward 90°: After ten turns to the left there follows a rotary nystagmus to the right, lasting for 11 seconds.

After ten turns to the right there follows a rotary nystagmus to the left, lasting for 8 seconds.

Caloric test: After syringing the left ear with 2700 c.c. of water at 55,° lasting over a period of 7 minutes, no nystagmus occurred in any position of the head, nor did the patient experience any vertigo.

SUMMARY.

1. The diagnosis of neurolabyrinthitis is established on the strength of the history and findings, particularly the latter. The impairment of hearing was distinctly of the perceptive type: He claims to have heard his own voice as though it was far away, and when he spoke his voice was rather loud, just the contrary to that found in the conductive type of deafness. Furthermore, the Weber was referred to the better-hearing ear; the bone conduction was shortened; there was diminution of hearing for low, middle and *high* tones by air conduction.

The vestibular nerve was involved along with the cochlear, as evidenced by the history of vertigo, the diminution of after-turning nystagmus to both sides, more especially to the affected (left) side, the negative caloric reaction, and the typical galvanic findings of vestibular nerve involvement, to-wit: Absence of reaction on the

affected (left) side, with preservation of reaction on the other (right) side.

2. In this case there were two etiologic factors present, either of which is capable of producing neurolabyrinthitis. They were syphilis and focal infection. In the case of syphilis, the tendency of the affection is to be bilateral; whereas, in focal infection the tendency is toward unilateral involvement. A knowledge of these facts led the writer to suspect the neurolabyrinthitis in this case to be of focal infection origin rather than syphilitic; however, it did not allow him to ignore the syphilis or to relax its treatment.

The characteristic blood findings clinched the diagnosis of focal infection. It has been the writer's experience, in cases of focal infection, to find the total white blood cell count ranging from 10,500 to 16,000 with a small lymphocyte count somewhere between 30 and 38 per cent. Even more conclusive were the blood findings on three consecutive days (March 18-19-20); the first count was made just before extraction of the last four infected teeth; the second count was made the day after, and the third count the second day after the extractions. There was nothing else done at this same time to influence the blood findings, nor did the character of the Wassermann change in any way.

The improvement in the blood findings marked a definite change for the better: *a.* general sense of well being; *b.* increase in body weight; *c.* cessation of vertigo; and *d.* rapid improvement in hearing.

Concerning the left vestibular *nerve* which was completely out of function at the height of his trouble, as indicated by its negative conductivity (reactibility) to galvanism, it improved sufficiently, after a few months, to be again responsive, but not so the vestibular *labyrinth,* which failed to react to cold water (caloric test).

The lack of improvement in his Wassermann, though unfortunate in a way, served a scientific purpose, and allowed us an opportunity to study the comparative effects of syphilis and focal infection under the most favorable circumstances. Had the syphilis played the least etiologic role in the neurolabyrinthitis, such marked improvement in every respect could hardly have been expected without a concomitant improvement in the Wassermann.

There can be no doubt as to the eventual success in the treatment of the patient's syphilis, since he is in the hands of two expert syphilologists, Dr. Strohm and Dr. Ludy, who have collaborated in the treatment of this phase of the case.

The patient still has tonsils which appear more infected than the average case calling for removal. They are to be removed at an early date by a capable nose and throat specialist of his own city, for fear that sooner or later they may serve as a focus for a recurrence of his neurolabyrinthitis.

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THE EMBRYOLOGY, ANATOMY AND PHYSIOLOGY OF THE ETHMOID AND SPHENOID SINUSES.*

DR. F. E. LEJEUNE, New Orleans, La.

In the study of the paranasal sinuses we find evidences in the second month of embryonal life of the development of folds and grooves on the lateral nasal walls. By the end of the third month these folds or turbinate bodies have assumed a definite shape and their corresponding grooves or meatuses are well outlined. All ethmoidal cells, the frontal sinuses and the maxillary sinuses have their origin from these preformed grooves or furrows between the folds which develop on the lateral nasal walls.

Davis in his extensive study of a sixty-day fetus, showed the development of a small projection under the middle portion of the middle fold or turbinate body, which in turn developed from the lateral nasal wall and proved to be the early bulla ethmoidalis. Just below this is found a narrow depression, the primitive hiatus semilunaris, separating the bulla from a ridge of mesenchyme representing the early formation of the processus uncinatus. From this it can be seen that the middle meatus early becomes and remains the most complex portion of the nasal cavity, particularly when we realize that from it develop the anterior ethmoidal cells, the maxillary sinuses and the frontal sinuses.

The ethmoidal cells are early divided into an anterior and a posterior group. All ethmoidal cells having their ostia inferior to the middle turbinate belong to the anterior group, while those cells having their ostia superior to the middle turbinate, regardless of their extent, belong to the posterior group.

Shaeffer tells us that the primitive ethmoidal cells develop as extensions of the nasal mucosa from the middle, superior and supreme meatuses extending into the surrounding areas. These extensions or invaginations representing the primitive ethmoidal cells appear in the fourth month of fetal life. By the seventh month these invaginations have become hollow, tubular or cylindrical processes and now appear as distinct cell formations. The anterior group of ethmoid cells appear to develop earlier and more rapidly than those of the posterior group and always arise from the middle meatus. The posterior group develop from the superior and first

*Read before the Symposium on Sphenoiditis given at the New Orleans Ophthalmological and Otolaryngological Society.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication August 23, 1926.

supreme meatuses and communicate with the nasal fossa above attached border of middle turbinate.

It should be recalled that the first supreme meatus is present in 60 per cent of adult nasal fossa and that in 75 per cent of cases a posterior ethmoidal cell communicates with it, indicating, therefore, that the ethmoidal cell in question arose from the first supreme meatus.

The ethmoidal cells are present at birth and continue their development until the age of puberty is reached. Their growth and development, according to Shea, is greatly influenced by any infections which they may suffer. There is no constancy as to number, position or size of the cells in either group. They vary in number from two to eight in the anterior group, while the posterior group may have from one to seven.

Differing in development from the early ethmoids, we find, about the fourth month of fetal life, evidences of the presence of the terminal nasal sinus. This, strictly speaking, is the primitive sphenoidal sinus. Unlike other accessory sinuses which develop from furrows or grooves located on the lateral nasal walls, the sphenoid develops as an invagination extending from the posterosuperior portion of the recessus ethmoidalis. The proximal end remains as the ostium of the sinus. Thus the primitive sphenoidal sinus comes to lie posterior to the nasal capsule and anterolateral to the body of the sphenoid bone, clearly showing that no portion of the fetal sphenoid sinus is contained within the sphenoid bone. A center of ossification known as the ossiculum of Bertini, develops in the anterolateral wall of this primitive sinus about the fifth month, and while it is well advanced at birth, it is not attached and does not become a part of the body of the sphenoid bone until the second or third year. The process of pneumatization occurs, involving the body of the sphenoid, and the results are the production of a definite sphenoidal sinus. Shea states that the sphenoidal sinus is generally a distinct cell at birth, but has no clinical significance, while Shaeffer claims that the sphenoid is early of importance clinically and that by the second or third year it has developed sufficiently to become the seat of pathological processes. From this time on the sphenoid develops rapidly so that by the age of 10 or 12 years it has attained its adult size.

The ethmoid labyrinth lies between the nasal cavities and the orbit, the frontal and the sphenoidal sinuses and between the floor of the cranial cavity and the middle turbinate. Unfortunately for the surgeon, these cells do not all remain within these anatomical limits. It is not unusual to find extensions of these cells into the middle turbinate, the uncinate process and more rarely into the neighboring bones

where the clinical symptoms simulate those of a maxillary or a sphenoidal sinusitis. The location of some of the anterior ethmoidal cells are such that they drain directly into the infundibulum ethmoidale, and from there, through the natural opening into the maxillary sinus, converting this cavity into a reservoir for infectious material. In those cases of chronic ethmoiditis in which all intranasal surgical procedures have failed and we are forced to resort to an external operation, we frequently find ethmoidal cells extending over and to the lateral side of the orbit. The position of these cells are such that no amount of intranasal surgery could reach them.

The proximity of the ethmoid cells to the orbit and the extreme delicacy of the lamina papyracea may permit the extension of an infection giving rise to an orbital cellulitis. The very intimate relationship of the cranial cavity by means of the lamina cribrosa and the possibility of the extension of the infection by way of the ethmoidal veins is always a source of constant fear. The ophthalmic veins may convey infection into the cavernous sinus, while the small vein which crosses the foramen caecum may infect the superior sagittal or longitudinal sinus. The anterior ethmoidal cells are in direct relation with the lacrymal fossa and sac and it is not uncommon in infections of these cells to see a secondary involvement of the lacrymal sac producing a dacrocystitis. The posterior ethmoidal cells, as well as the sphenoidal sinus, come into very close relationship with the optic nerve as it approaches the orbital apex and passes through the optic foramen. The exact relationship will vary with the size, shape, symmetry and location of the sinuses. The optic commissure very often is situated directly over the sphenoidal sinus, so that any infection arising in these sinuses may readily affect the nerve in this location (Gottlieb). The cavernous sinus with its contained structures, the internal carotid artery, the abducens, trochlear oculomotor, ophthalmic and maxillary nerves, lie to the lateral aspect of the sphenoids. The hypophysis cerebri lies in a depression over the body of the sphenoid, while in the floor we find the vidian nerve emerging from the pterygoid canal and the maxillary traversing the foramen rotundum.

The physiological function of the accessory sinuses at best are very obscure. It is thought the size of the sinuses may have a direct influence on the resonance of the voice. The theory generally accepted is that they play an important role as an aid to respiration, particularly in warming and humidifying inspired air. This being true, there must be a constant flow of a column of air through the paranasal sinuses with every inspiration and expiration maintaining proper ventilation and facilitating drainage. It is obvious that any

pathological condition interfering with the normal ostia of these sinuses must correspondingly interfere with their function. The extent to which this takes place depends greatly upon the size of the ostia of the different sinuses.

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DEFECTS OF SPEECH.*

DR. WILLIAM J. PEPARD, Philadelphia.

In working with people who are troubled with defects in speech I usually classify them into groups according to their specific difficulty. One group consists of stammering and stuttering, another lisping, some are lallers, defective phonation, foreign accent, and then there are those who cannot talk at all.

It is convenient to classify them thus for the means employed in correcting these various forms of defects are definitely formulated.

A stammerer, for instance, has to be handled differently from a lisper, a lisper differently from one who has some form of defective phonation, and, again, a person who wishes to have a foreign accent corrected is still another proposition.

The problem of developing speech in a speechless child is one that is governed largely by the individual. What will be effective with one, may prove useless with another, but there is nearly always some key to the solution of the trouble.

I usually employ another classification of all of these conditions, namely, those of a more physical nature and those of a more psychic nature.

Quite frequently these speech cases have physical defects and malformations accompanying them, such as adenoids, hypertrophied tonsils, deflected septum, tongue tie, etc. It is here that the work of the laryngologist must contribute to the correction, as it is always desir-

*Read before the Philadelphia Laryngological Society May 4, 1926.
Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication June 28, 1926.

able to correct these physical handicaps first in working for better speech.

If, however, a child has already acquired an incorrect manner of producing speech sounds, the removal of the contributory physical handicap will not correct the speech.

The element of habit will cause the person to go on producing the sound or words as before. This habit must be destroyed and the correct habit established. Many doctors still tell people that when the tonsils are removed the patient will talk allright. A child was brought to me stammering badly. The mother told me that her doctor said to have his tonsils removed and he would outgrow his trouble. I do not believe that any case ever outgrows a speech defect. It is undoubtedly true that some persons through superior mentality, perhaps, are able to eradicate their difficulty through self-administered correction, but the great number of those who are not able to do so establishes the fact that it is not safe to expect it.

I have seen some cases where the soft palate fell down after a tonsillectomy. Due, perhaps, to some injury to the muscles supporting the palatal arch, such as the pillars of fauces. The malposition was so acute that all attempts at speech were being forced through the naris, making the child sound very unintelligible and idiotic. This can be corrected by a certain simple exercise which tends to strengthen the muscles and to restore a more normal formation of the palatal arch, thereby permitting the vocal sounds to pass through the buccal cavity instead of being checked by the occlusion formed by the soft palate lying on the rear of the tongue.

In the group which I have termed speechlessness, there are many cases that are being neglected entirely because it is thought that nothing can be done for them. Cases, for instance, of infantile paralyses, and certain types of injury to the mechanism involved in speech, existing from birth or perhaps from before birth, and also retardation of a complete mental development. This latter form I prefer to consider as arrested mental development due to a failure of the speech mechanism to commence functioning.

Unfortunately, these cases are usually considered helpless and nothing is attempted to improve their speech. No matter how severe the case may look, or what the cause of the trouble may have been, every person who has trouble with speech should be given the help of someone qualified to develop their speech abilities to the best possible degree.

Speech is undoubtedly the most potent factor in developing the human being to a complete and successful life, and is the one faculty that differentiates him completely from others of the higher animals.

Orthopedic Hospital.

FRONTAL SINUS DISEASE: THREE UNUSUAL CONSECUTIVE CASES.

DR. LEE M. HURD, New York.

Case 1: Acute Purulent Infection Probably Associated with Mucoceles from the Beginning.

Dec. 4, 1906, boy, age about 16 years. Severe pain and tenderness over left frontal sinus for two days. Temperature 105,° pulse 135, following a chill in the morning. He has had recurrent pain in this region accompanying coryza for several years.

Examination of nose: congested and inflamed mucosa about anterior portion of middle meatus with some pus. Right frontal transilluminates clear, left dark, antra are clear. Enlarged tender gland at angle of jaw left.

Blood count showed 3,000,000 reds, 25,500 whites, 81.6 per cent polynuclears, hemoglobin 88 per cent, no malaria plasmodia found. Ordered hot saline douches every hour. The temperature the next day was 99⅘° at 3 a. m., 105° at 3:30 p. m. I removed the anterior portion of middle turbinate, tried to douche frontal sinus but could only get into a large ethmoidal cell. Second day, temperature again reached 105,° pulse 135.

I then decided to open externally via Killian route; found the frontal sinus full of pus under pressure, lining mucosa was necrotic and was removed in toto. The sinus was large for his age, extending beyond supraorbital notch, and 1 inch in height, with deep orbital prolongation. I could find no opening into nose. A large anterior ethmoidal cell seemed to have expanded and blocked the frontal orifice. This ethmoidal cell had a very thin bony wall and was filled with a thick mucoid material. The anterior ethmoidal cells (which were congested, but showed no pus) were removed. The wound was closed. That night temperature was 103°; the next day 105,° then dropped to 100,° and normal on the third day. Four weeks later interior of nose was entirely healed.

Nov. 16, 1908: I saw him again with history of progressive swelling for a period of two weeks over central frontal region and which was about 2 inches in diameter and fluctuating; also a second swelling over the old wound of left frontal sinus with much edema of both upper lids and also edema of the scalp back to the occiput. An incision 1 inch long was made through the inner part of old

*Read before the Forty-eighth Annual Meeting of the American Laryngological Association, Montreal, June 3, 1926.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication June 4, 1926.

scar and about 1 ounce of pus evacuated. The left or previously infected sinus was filled with pus; the posterior wall was covered with dense fibrous tissue, and the interfrontal partition was necrotic and perforated; the right frontal sinus walls were covered with thick polypoid membrane and the nasal orifice of the right sinus was situated rather posteriorly and was small.

Two weeks later when the edema and swelling had subsided, through a double Killian incision the floor and the balance of the interfrontal partition and all the frontal mucosa was removed, but the anterior frontal wall was preserved and the right ethmoidal cells exenterated. On the left side the dense fibrous tissue completely closed off the left nasal cavity. A pure culture of long chain streptococci was obtained from the pus.

In 1912 a small movable, painless, elastic mass could be felt behind the nasofrontal articulation where the floors of the frontal and ethmoids had been removed. In September, 1913, he had a coryza following surf bathing; there was a feeling of fullness about right frontal with some purulent nasal discharge; he was tender over right frontal and very tender to inner side of the right eye in ethmoidal region. Suction brought nothing from the frontal region. The temperature was normal.

In June, 1921, the swelling behind bridge of nose was larger and a protrusion of right eyeball, which had been present for some months, had increased.

In September, 1922, swelling behind bridge of nose was larger, and eyeball had markedly protruded downward and outward.

In December, 1922, orbital swelling, pain, edema of lids with congested conjunctiva and proptosis occurred. Pain and swelling increased. A 1-inch incision was made in right scar, evacuating 2 drams of very thick mucopus (staphylococcus pyogenous aureus). Four days later eyeball had receded 50 per cent; there was some diplopia.

Jan. 9, 1923: Under general anesthesia I opened through previous double incision and found large cyst or mucocele in upper part of orbit and right frontal sinus, which had by pressure doubled the size of sinus cavity. The contents of the cyst was thick mucus, and the lining membrane very thin.

The mass previously mentioned, in the median line behind the nasal bones, proved to be another cyst. Three other small cysts behind this one and to the left were removed. Large openings were made into both sides of the nose. All of the interfrontal septum was removed with part of the nasal septum to the crista galli. On Feb. 23, the right eye was reported normal.

As it was impossible to be sure that all cystic membrane had been removed, the Lothrop technic of drainage into the nose was done. Since 1923 he has had a thick, viscid, orange-colored discharge into the nose from time to time, and although I have douched the frontal cavity many times, I have never recovered any of the cyst secretion.

Case 2: A Condition of Ossifying Periostitis.

A man, age 43 years, had had trouble with nose, and frontal headaches for twelve years. An external frontal and antral operation, right side, was done nine months ago, since which time he has had repeated attacks of frontal inflammation with purulent discharge through fistula in upper lid. Reoperated a few weeks ago, no better; purulent discharge from nose, no headaches since last operation.

My examination showed pus from right frontal into nose and from fistula through scar in right eyebrow. Transilluminates dark for all sinuses. Large, soft mass in region of right ethmoids. Douched pus from right frontal sinus via fistula. Antral douche showed only a few shreds. Four days later only got few shreds on douching frontal. Fistula closed. Two days later, tender swelling right forehead, pus from frontal sinus into nose.

Operation: Opened right frontal, usual incision, anterior wall entirely removed and found this was greatly thickened with new-formed bone; there was as well a thickening of posterior wall, so that the greater portion of sinus cavity was represented by a cleft not more than 2 m.m. wide, except at the lower inner portion, where there was a small cavity about 5x10 m.m.; the anterior wall was from 10 to 15 m.m. thick. All this new-formed and softened bone was removed. The ethmoidal cells were filled with polyps and were exenterated. Antrum was opened through canine fossa and cavity found almost obliterated with fibrous tissue; he was entirely healed in eighteen days except for some crusting in nose.

Case 3: Acute Panostitis Following Trauma from Impact with Water with Probable Rupture of Orbital Ethmoidal Plate.

A girl, age 18 years, who had been under my observation for the previous eight years, and who at the age of 10 years had an acute antrum on the right side complicating influenza and no other trouble with the nasal sinuses until she fell from an aquaplane, striking the water with considerable force; that night she had pain about the left eye and swelling of the lids rapidly developed. When I first saw her five days later, she had swelling of the eyelids, forehead and cheek, with congestion of the conjunctiva and pain in eye. The eyelids

were so swollen she could not open the eye; she also had tenderness of the frontal sinus; temperature 102.°

I immediately operated via Killian route; the mucous membrane of left frontal sinus was loose and gangrenous and lifted out in toto; the interfrontal partition was red, softened and perforated, with free pus in right frontal sinus; ethmoid cells full of pus, and the membrane, like the frontal, could be lifted out in toto; and the bony partitions of the ethmoids were breaking down.

The periorbita in relation to the anterior ethmoid cells was covered with fibrin and thickened. The posterior ethmoid cells were not affected.

I removed the anterior wall, the floor of the frontal sinus and all of the interfrontal partition, and removed the bone between the right frontal orifice and the left side (a modified Lothrop technic) and a portion of the nasal septum was removed just below the floor of the two frontals.

I also removed all the anterior ethmoidal cells and explored the posterior cells and incised the periorbita through the thickened portion, but did not find pus; closed incision with small drain at internal end. Next day there was much less swelling of eyelids and forehead, and some pus from drain; two days later considerable pus through drain and into nose; temperature 101.° Five days after operation all stitches were taken out and wound strapped with adhesive, and dressings left off. Considerable pus in nose; irrigating with Dakin's solution. An external strabismus due to paresis of the internal rectus which had been present before the operation had nearly disappeared on the fifteenth day. Almost no pus from nose, but bare bone could be detected on posterior wall and bridge of frontal sinus and roof of ethmoids and through a small fistula at lower end of incision the bony bridge seemed movable. Twenty-five days after first operation I again opened through first incision and found the bony bridge, the entire posterior wall of frontal sinus and the cribriform plate necrotic; this necrotic bone was removed in several large sequestra, revealing granulations on the dura; the wound was sutured and I depended on drainage through the nose. The sutures and dressing were removed on the second day, and on the fifth she returned home. The diplopia disappeared in a few weeks and she has remained well a period of two and a half years, except for an acute antrum on the opposite side, six months after the operation.

39 East 50th Street.

ABSCESSES OF THE LEFT FRONTAL AND TEMPORAL LOBES, OF METASTATIC ORIGIN.

DR. PAUL L. MAHONEY, Little Rock, Ark.

This case was of special interest to me, for at no time was there any definite symptoms upon which a diagnosis of our final findings could have been based.

While I feel that nothing was left undone, the fact remains that early diagnosis and properly instituted treatment is, after all, the prime factor in the treatment of these conditions.

J. C., age 42 years, white, male, came to our office Aug. 2, 1925, complaining of coryza, headache and sore throat of several days' duration. General health up until this time had been good.

Family history of no importance.

Bilateral chronic otitis media since early childhood. There was no history of any serious illnesses in the past.

Ears: Membrana flaccida with portion of malleus was present in right. Scant, nonodorous, dirty brown discharge. Left ear showed drum absent in its entirety and scant, odorous discharge typical of bone invasion. There were no polypi present in either ear. Hearing in both was diminished—markedly so in the left. The usual tests for hearing were carried out with the expected findings. Fistula—caloric plus.

Nose: Slight deviation of septum with ridge upon the left. The usual findings of hyperesthetic rhinitis were present. There was no free pus or evidence of hyperplastic sinusitis.

Teeth fair, tonsils showed subacute inflammation, associated with a chronic lateral pharyngitis. The post-nasal space showed slight swelling of tubal tonsils, the mucous membrane being darker red and more moist in this locality.

Eyes: While vision had always been poor in left, no evidence of pathology was noted in either. Vision corrected to normal in right. He was placed upon routine treatment and asked to return.

Aug. 4, 1925: Several days later I was called by his physician to see the patient at his residence. The doctor reported that general physical examination was negative, and thought the slight elevation of temperature that was at this time present due to a focus of infection in his throat.

While I found the condition of his nose and throat unchanged, he was complaining of a more intense headache, and tenderness in

Editor's Note. This ms. received in The Laryngoscope Office and accepted for publication July 23, 1926.

region of sternomastoid muscle. There was no nausea, vomiting, nystagmus and all reflexes were normal. His condition remained stationary for three days.

Aug. 7, 1925, I was called to see him at 11:30 p. m. He was extremely nervous, mentally confused and continually placing his hands to his head. Temperature 101,° pulse 84. I immediately had him admitted to the hospital, where various consultations were obtained.

X-ray showed no breaking down of bone, but sclerosis on both sides: Blood: leucocytes, 20,000; small lymphs., 15; large lymphs., 1; polys., 84. Widal, Wassermann and malaria negative.

Spinal fluid: C. m.m. 200-70 per cent lymphocytes. Cultures were sterile, and guinea pig inoculation failed to isolate the T. B. bacillus.

Urine: Negative.

Temperature ranged from 101-102,° pulse 80-90, respiration 20-24. All reflexes remained normal and mental confusion gradually improved. There was still no nausea, vomiting, dizziness, but continued impairment of appetite, emaciation, prostration and headache. The latter was the only symptom present of cerebral pressure.

Aug. 15, 1925. Patient this date complained of slight nausea, and his general condition remained unchanged. While we thought of many possibilities and were not certain of any, we decided to and the next day did a radical mastoidectomy on the left side.

Findings: The bone was greatly ebonized and hemorrhagic. Several drops of pus were found in the region of the antrum. Organism was streptococcus. Bone in region of aditus showed some necrosis. There was no evidence of destruction in region of tegmen antri or tympani, although there was a small amount of cholesteomatous material present.

Aug. 15, 1925: There was an evident incomplete facial paralysis this date. Temperature 100-102,° pulse 78-90. He was very optimistic, and was anxious to learn just how long it would be before he could leave the hospital. His only complaint was slight headache. Mental faculties were very clear.

Aug. 19, 1925: Headache not severe, but early in the afternoon he had a severe chill, followed by typical symptoms of a lateral sinus thrombosis. He did not complain of any pain in the region of the left ear.

Aug. 20, 1925: Was operated this morning. The sinus was deeply placed and bleeding was very annoying. The sinus wall was discolored and aspiration revealed a thick, cloudy serum. The vein was opened and revealed a dissolution of a thrombus. Attempts to re-establish circulation from either end failed, and we ligated the jugular vein.

Aug. 21, 1925: Temperature 102,° pulse 88, remaining so for several days. Facial paralysis still incomplete, but no other abnormal findings.

Aug. 23, 1925: There was some difficulty in speech today. This greatly worried him and he continually smoked cigarettes. His condition gradually became worse and difficulty of speech resulted in complete loss. At this time there was some difficulty in using his right arm.

Aug. 25, 1925: Was given a transfusion today with the hope that it might better his condition. There was very little reaction.

Aug. 25, 1925: Has become very restless; there was slight mental confusion, but he continued to take a little nourishment.

Aug. 28, 1925. Restlessness more pronounced and was beginning to have slight rigors. Temperature ranged from normal to 101,° pulse 74-110, respiration 20-30.

Aug. 29, 1925: Perspired freely all day, and had several convulsions. Paralysis in right arm complete, with evident bulbar paralysis. It was becoming necessary to administer narcotics at shorter intervals. Temperature and pulse began to climb, patient became comatose and general condition grew worse until death.

Autopsy: The dura was thickened, reddened and somewhat discolored over the anterior pole of the left frontal lobe. It was not adhered to the bony cavity. It stripped easily from the cortex, except over the anterior lobe of the left hemisphere. On removing this a purulent discharge was seen over the anterior pole, involving the second third frontal lobes, with a slight involvement of the first. Further investigation revealed numerous abscesses located throughout the frontal lobe, especially in the second and third convolutions. In these convolutions old blood clots had begun to disorganize. These evidently formed at the beginning of the infection.

On sectioning the temporal lobe, a large abscess the size of a large pecan with a well organized sac was found. This corresponding to the region of the tegmen tympani, probably the source of the infection. The occipital and parietal lobes, cerebellum, medulla and pons were not involved. They showed a slight congestion, which was secondary to the infection, but no exudate, transudate or abscesses were seen. The ventricles were not enlarged. The spinal fluid was clear. There was no sign of any inflammatory condition found in the ventricles.

Diagnosis: It is my opinion that the abscess in the frontal lobe was the first to form because of the size, the organized capsule, and further changes than found in the other abscesses. I believe that these abscesses were of several months' duration.

418-22 Exchange National Bank Building.

DIFFERENTIAL DIAGNOSIS AND TREATMENT IN DIPHTHERIA.*

DR. H. HUNTER LOTT, Philadelphia.

To differentiate diphtheria from other conditions with which it may be confused, let us first consider the things it mostly resembles or which mostly resemble diphtheria upon inspection.

One very well known author states (Diseases of Nose, Throat and Ear, Ballinger, p. 485) the differential diagnosis of diphtheria should be made between *a.* peritonsillar abscess; *b.* follicular tonsillitis; *c.* pseudo diphtheria; *d.* pseudo croup; *e.* catarrhal rhinitis. The chief diagnostic points are the microscopic and the culture findings.

Another states that Vincent's angina is to be differentiated from diphtheria, and still others do not seem to think diphtheria need be differentiated from anything.

Data by Dr. C. Y. white, of the City Laboratory, shows there must be some conditions that should be differentiated from diphtheria, inasmuch as there were 12,004 cultures made during 1925 in Philadelphia, and only 2,359 were positive on first culture. Of course, those figures don't mean a great deal, because so many cultures are made to detect a carrier or from persons who are not ill, but had been exposed.

Dr. White also states the predominance of organisms other than diphtheria are: *1.* Staphylococcus. *2.* Mixed growth of mouth organism. *3.* Streptococcus, long and short chain.

It would, therefore, seem in Philadelphia, at least, that acute follicular tonsillitis is the most frequent condition confused with diphtheria.

Let us consider the symptoms of each:

	Diphtheria.	Acute Follicular Tonsillitis.
1.	Onset temp. rise gradual.	Sharp and rapid.
2.	Slow, feeble pulse.	Rapid, bounding pulse.
3.	Depression marked.	Slight depression.
4.	Exudate extends beyond tonsil to surrounding parts.	Exudate on tonsil only.
5.	Exudate firm and tenacious.	Soft and friable.
6.	Exudate closely adherent.	Not adherent.
7.	Exudate membranous and can be removed in strips.	Not membranous.
8.	Raw, bleeding surface under membrane.	No bleeding surface.
9.	Cervical glands swollen and tender, even in mild cases.	Glands not swollen, except severe cases.
10.	Vomiting and delirium.	No delirium.
11.	Albuminaturia common.	Albuminaturia occasioned.
12.	Klebs-Loeffler's bacillus present.	Klebs-Loeffler's bacillus present.

*Read before the Philadelphia Laryngological Society.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication June 28, 1926.

Of course in making a diagnosis today, a laboratory is always available and a smear and culture are readily made. If the laboratory report is positive the diagnosis is no longer in doubt, but it is when the laboratory report is negative and the symptoms are positive that one is confused. For example, I will cite a case seen recently of severe toxic symptoms, sore throat and tender, swollen glands in the neck, of sudden onset and rapid development.

M. F., child, age 5 years. First saw her Jan. 22, 1926. Complained of sore throat, pain and stiffness in neck. Illness began twelve hours previous and continued to progress. Child was restless, pale, no vomiting nor nausea. Pulse 140. Temperature 102° at night.

Inspection of throat: Congestion of pharynx with discreet patches of exudate, grayish in character; tongue coated and swollen. Tonsils inflamed, but no exudate; glands in neck swollen and tender. Nasal discharge, yellowish and profuse. Twenty-four hours later, all symptoms worse. Temperature 102° morning, 103.5° night. Pulse 140-60. Albuminaturia.

Child more toxic, some delirium. Exudate had coalesced and was now a grayish white smear over entire surface of pharynx and forward to posterior pillar of tonsils. Culture negative for diphtheria. Streptococci growth positive.

Twenty-four hours later, continuance of symptoms, temperature 104.3,° pulse 160. Delirium more marked, nasal discharge thick, dark and viscid, exudate over surface of tonsils, marked depression, child looked very ill. Culture from throat negative for diphtheria. Blood culture report at this time: hemoglobin, 85 per cent; red count, 3,550,000; white count, 15,600; differential: polyp., 58; s. lymph., 31; l. lymph., 9; trans., 0; mono., 0; baso., 0; eosin., 2. Blood culture, sterile forty-eight hours. Throat culture, hemolytic streptococci and staphylococcus aureus.

When this child was first seen a diagnosis of streptococcus pharyngitis was made. Silver nitrate, 20 per cent, was applied to the patches of exudate on pharyngeal wall, and 20 per cent argyrol dropped into nose every four hours. Heat applied to neck and free purgation. Second day, 50 per cent silver nitrate was used and streptococcic vaccine give hypodermically, ⅕ c.c. of 100,000,000 to cubic centimeter.

Third day, 10,000 units diphtheria antitoxin given, followed by marked and rapid recovery.

This case was never demonstrated to be anything but a severe streptococcic infection and yet there was a most marked improvement when antitoxin was administered.

Vincent's angina may at times resemble diphtheria, especially if it be of long standing, with mixed infection and widespread ulceration. The exudate forming on the ulcerated area resembles a membrane and at times there is great depression and toxic symptoms. Seen later, however, the history shows a longer duration than one would find in diphtheria and if watched for a day or two the exudate and ulceration will be noted to develop very slowly, whereas if it were diphtheria, there would be, in twenty-four hours, with such a free start, a marked increase in all symptoms and a rapid spread of the membrane.

In making an examination of suspected diphtheria case the laryngeal mirror and Jackson's laryngoscope should be employed, care of course being taken to prevent the patient coughing in one's mouth or eyes. In making smears from the throat it is well to include nasal and laryngeal smears. One negative smear should not be final if symptoms do not abate.

In *laryngeal diphtheria* there is hoarseness and a high pitched, metallic cough, the so-called croupy cough, which comes on in paroxysms. There is a slight rise in temperature and the frequency of respiration is slightly increased. As the exudate extends, the hoarseness and aphonia increase. Finally, the respiration becomes embarrassed and stridulous. The auxiliary muscles of respiration are brought into action. There is marked retraction in the supraclavicular and suprasternal spaces; also at the substernal space and at the border of the ribs. The alae of the nose dilate with each respiration. The inspiration is long, deep and labored and more difficult than expiration, which may be comparatively easy. The child is restless, clutching at the sides of the bed or anything to raise itself up. The face is pale and bathed in a profuse perspiration. The patient has a wild, hunted expression. As the obstruction increases, cyanosis appears, the extremities become purple, the lips and face of a livid hue. Sometimes during a fit of coughing, membrane is expelled as a complete cast of the larynx, trachea, and sometimes even of the smaller bronchi. This, as a rule, only gives temporary relief, for the membrane quickly reforms and all the symptoms return. Unless these cases be relieved by intubation or tracheotomy, cyanosis becomes greater, until the child dies, asphyxiated.

There is still a diversity of opinion as to the treatment, both as to the local and general symptoms, but the ones who know best—the ones who have had the greatest experience, those who are and have been connected with municipal hospitals for contagious diseases, agree upon one thing: the early and free use of antitoxin, even though they disagree about the method of administration.

Woody (Tice's Practice of Medicine, Vol. 3, p. 131), who has had a vast and varied experience, recommends in a case of tonsillar type with limited membrane 10,000 units, if given the first day of the disease and there being no signs of dyspnea, if there be dyspnea, 15,000 units. If the case is seen on the second day, 15,000 to 20,000 units. In nasal cases with slight discharge he uses the same doses, but in nasal cases showing a distinct membrane and discharge and signs of toxemia he uses 20,000 on first day, increasing to 30,000 or 50,000 if treated as late as third day.

If the antitoxin be used intravenously, 3,000 to 5,000 units are given in place of 15,000 to 20,000 doses subcutaneously.

Some authorities think that while one dose is sufficient, the second dose is to be recommended, if at the end of twelve or twenty-four hours there be no cessation of symptoms and the membrane continues to develop; while others claim the second dose is of no value. It seems to me the former method is the more logical and that good is derived from the second injection, even if not as great as the first.

One thing that seems to be agreed upon is the early administration of large doses of antitoxin, even in doubtful cases where a membrane or exudate is not shown to be diphtheritic by smear and where the patient has toxic symptoms an early dose of antitoxin will at least do no harm, but is followed by the most gratifying relief of symptoms. This applies, in my judgment, to a severe acute follicular tonsillitis and more especially to a streptococcic infection.

Locally various washes, gargles and douches have been suggested. It is doubtful if there is much to be accomplished except as mechanical cleansing of the parts. Mild solutions directed toward stimulation, as boracic acid, chlorid of sodium, etc., at a temperature of 110,° are used in fountain syringe placed about 1 foot above head of patient.

General treatment consists in adminstration of stimulants to overcome depression, the weak heart, irregular pulse and septic condition.

Whisky and strychnin are beneficial and free elimination should be maintained.

If laryngeal symptoms progress, intubation or tracheotomy must be resorted to.

1530 Locust Street.

INTUBATION AND TRACHEOTOMY IN DIPHTHERIA.*

DR. ARTHUR J. WAGERS, Philadelphia.

What is the condition arising during the course of illness from diphtheria which renders the operation of either intubation or tracheotomy advisable, if not actually imperative?

In a word, it is partial occlusion of the airway through the larynx, which, if not promptly relieved will shortly end in complete occlusion and death from asphyxiation. The principal cause of this obstruction in diphtheria is the presence of diphtheritic membrane, combined with a certain amount of edematous swelling of the mucosa.

These obstructive factors generally appear in those cases in which the disease has not been recognized sufficiently early and the use of antitoxin has been too long delayed. We recognize this condition of laryngeal obstruction by the distinct effort the patient makes to breathe—the suprasternal and intercostal indrawing—the increasing restlessness, up to the point of exhaustion, when the child may become very quiet. This quietness should not be interpreted as a favorable sign in diphtheria, particularly when occurring with cyanosis and a failing pulse. Under such circumstances death is approaching and only quick and decisive action will prevent the otherwise inevitable. Which, then, is the operation of selection?

In answering this question we are obliged to give consideration to the varying circumstances of location of patient with reference to accessibility to contagious disease hospitals, to the instrumental equipment of the attending physician, and to his experience and skill, or lack of experience and skill, in the performance of the operative procedures under discussion.

In any large city having a hospital for contagious diseases, it is a comparatively simple matter for the attending physician, whose experience with diphtheria may be very limited, to notify the bureau of health that he has a case of suspected diphtheria. If he wishes, an expert diagnostician will be sent at once, and if the diagnosis be confirmed, the ambulance is called, and the child taken to the city institution, where it comes under the care of trained experts, who have every facility at hand with which to meet any emergency which may arise.

It is quite a different matter for the attending physician practicing in the hundreds of communities which are remote from contagious

*Read before the Philadelphia Laryngological Society April 6, 1926.
Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication June 28, 1926.

disease hospitals. One should not for a moment underestimate the diagnostic skill or practical ability of these men in caring for the ordinary case of diphtheria. These men of the rural communities will make just as accurate diagnoses and administer antitoxin just as promptly as the city practitioner, the only difference being that when emergencies arise, the former must be his own expert—he must meet conditions as they arise, however formidable they may be.

Because of the fact that antitoxin is administered early in the disease, laryngeal obstruction from the presence of membrane does not occur in the majority of all cases treated. Consequently, the general practitioner has but little opportunity to acquire skill in operative procedures from the cases treated in his private practice. Given, then, an instance in which relief from dyspnea is immediately indicated, and the attending physician having no personal experience with either intubation or tracheotomy, which would be the better operation for him under the circumstances? In the first place it may be noted that comparatively few physicians have an intubation set of instruments at hand. But even if our physician has the instruments and has never attempted an intubation, we believe it would be decidedly safer for his patient if he were to perform a tracheotomy. Intubation as pictured and described in the textbooks is rather a simple operation—practically, for the uninitiated, it is very difficult. And a dying child is in no condition to suffer the additional strain imposed on its weakened body by the prolonged efforts made to successfully introduce a tube into the larynx. On the other hand, while the same physician may not have at hand a tracheotomy tube, he can meet the indication if he must, with only a sharp knife, a few hemostats, and a cool head.

If the condition presented in any given case is not an actual emergency—if the dyspnea is observed early, and the medical attendant has the necessary instrumentation and time in which to prepare to meet the indications by either intubation or tracheotomy, which, then, would be the operation of choice?

Ordinarily, the establishment of sufficient airway would be accomplished by intubation. If the inflamed laryngeal mucosa is not too severely abused by the introduction of the tube, if the tube is of proper size, and if it is not too frequently coughed up, necessitating numerous reintubations, it will in many, if not most cases, carry the patient safely through the critical stage of laryngeal involvement in from four to six days and can then be permanently removed. If, as not infrequently happens, the tube is coughed up several times daily and as often replaced, and with perhaps substitution of a larger tube from time to time, the writer believes that intubation should be dis-

continued after a day or two of this kind of treatment, and tracheotomy performed. Although intubation may be skillfully done, it appears only reasonable that the manipulation incident to the introduction of the tube must cause a certain amount of trauma to the highly inflamed larynx and tend in time to bring about a stenosis of the larynx, which in the end means either prolonged wearing of a tube, or a tracheotomy and long, tedious laryngeal operation to overcome the stenosis. Far better to prevent this stenosis, if possible, than to treat it after it has occurred.

OPERATION.

Intubation: The O'Dwyer intubation set is the one most commonly used. It consists of a series of graduated tubes numbered according to the age of the child on which they are to be used, from 1 to 12 years; a side mouth gag, an introducer, and an extractor.

It is advisable to have instruments for doing a tracheotomy at hand before attempting intubation.

With the arms resting against the side of the body, the child should be closely wrapped in a sheet from the shoulders to the feet. It may be held in either the upright position or lying down, according to individual preference of the operator. The mouth being held open by the mouth gag, the operator introduces the index finger of the left hand until he is able to press the tip of the epiglottis against the base of the tongue. With this finger as a guide, he passes the tube with the introducer back over the median line of the tongue until he feels that the advancing end of the tube has just passed over the tip of the epiglottis. The handle of the introducer is then elevated until the proximal border of the tube can be felt hugging the posterior surface of the epiglottis. In this position, if the tube be of proper size it should pass readily downward through the glottis. The tube is held in position with the tip of the guiding finger while the introducer is removed. A cord should always be attached to the upper end of the tube, the free end being held with the handle of the introducer, the object being to make a hasty rescue of the tube in case it becomes detached from the introducer during the manipulation, or in case of need for hurried removal of the tube, after proper placement, it may be lifted out by means of the cord. In some instances this cord is removed after the tube has been successfully placed, in other cases it is allowed to remain, the free end being attached to the ear or side of the face.

Before resorting to any operative measure for the relief of dyspnea in these cases, it would be well, if the necessary apparatus be at hand, to make an effort at clearing the airway by means of suction. In some instances this may be sufficient to relieve the patient.

If so, it is a better method than either intubation or tracheotomy. In any case the lower pharynx and a portion of the larynx will be cleared of possible loosened patches of membrane mixed with a quantity of tenacious, thick, mucoid material, thus rendering intubation more certain of being effective if it must be done.

A successful intubation is recognized by the immediate relief of dyspnea. Should this not take place, it will probably be due to the fact that a fragment of membrane occludes the lower end of the tube, or else there is marked tracheal swelling below the tube, or, what frequently happens, the tube has been introduced into the esophagus instead of the larynx. If the difficulty be the presence of membrane, the tube should be removed at once, when, from coughing, the offending membrane may be dislodged and coughed up and the tube replaced. When satisfied in any case that intubation is not affording the relief sought, a tracheotomy is immediately indicated.

From the moment the tube is introduced, in an acute case, the patient should be under the watchful eye of a nurse, since there is always the possibility of the tube becoming blocked, or expelled while coughing, when its immediate replacement becomes necessary.

At the Philadelphia Hospital for Contagious Diseases, a separate building is maintained for intubated cases, having one floor for the acute cases and another for those cases in which it has become necessary to prolong the intubation after the acute condition has subsided. This building is equipped with a bell signal system by which the physician on duty is called at any moment should a tube become blocked or expelled.

It is evident that the management of intubated cases which must be cared for in the home becomes a matter of considerable concern to the attending physician, and unless he can arrange for emergency intubation, he would do well to perform tracheotomy at the outset.

Tracheotomy. The indications for tracheotomy are the same as for intubation. It is done to provide a clear airway to the lungs when the natural air passage through the larynx or upper portion of trachea is obstructed by disease. In the present instance we are considering laryngeal diphtheria, although the operation is performed in the same way and the after-care is the same regardless of the condition necessitating the operation. Mention has already been made of the essential instruments, a knife and hemostats, required for making a hasty opening of the trachea. It might be necessary in a given case to reduce this instrumentation to a knife only, and not a suitable one at that. However, if the case is seen sufficiently early, one should not await dire emergency before operating.

Having sufficient time in which to prepare for an orderly operation, the following instruments should be at hand: sand bag, hypodermic syringe with fine needle, sharp scalpel, hemostats, retractors, tenaculum, sharp pointed, curved bistoury, blunt pointed, curved bistoury, Trousseau dilator, tracheotomy cannula with pilot, scissors, ligatures and gauze sponges.

Operation. General anesthesia should never be employed. If the case is one of extreme emergency there is no time to apply local anesthesia and it is scarcely necessary since the patient, bordering on unconsciousness, will take but little, if any, note of what goes on about him.

When time permits, a series of intradermal injections of 1/10 of 1 per cent cocain solution is made in the midline of the neck from the thyroid cartilage to near the suprasternal notch. Each of these injections, if made within the skin and not subcutaneously, produces a rounded, white wheal.

With the patient lying on its back, a sand bag is so arranged beneath the shoulders and back of the neck, that the front of the neck is extended and somewhat elevated. Care should be taken that the head is held perfectly straight with the nose pointing to the zenith. Incision is made from the prominence of the thyroid cartilage downward, exactly in the median line, to a point just above the suprasternal notch. Having divided the skin, superficial fascia and muscular layers, the trachea should be exposed as far as possible by blunt dissection. Bleeding from the thyroid vessels, which may occur in spite of careful dissection, will be controlled by hemostats. If the isthmus of the thyroid interferes with exposure of the lower tracheal rings, it may be cut through and the two sides retracted along with the other tissues. With the upper four rings of the trachea exposed and all bleeding controlled, one is ready to incise the trachea. The tracheal wall should be steadied by means of a tenaculum and held to its natural position while the sharp pointed bistoury is inserted exactly in the midline between the fourth and fifth tracheal rings, and the fourth ring cut from below upward. The incision through the third and second rings may more safely be made with the blunt pointed bistoury. The first tracheal ring should not be cut. With the Trousseau dilator the tracheal incision is held open while the cannula with its pilot is inserted and the pilot immediately withdrawn. All bleeding having been controlled, the edges of the wound in the neck are simply brought together without suturing and a pad of gauze, moistened with 1-10,000 bichlorid solution, arranged around the cannula and beneath the tapes which are at-

tached to the cannula and tied about the neck to prevent the cannula from being ejected.

The operation described is commonly designated as "low tracheotomy". Dr. Chevalier Jackson, than whom there is no higher authority on the subject of tracheotomy, declares that the operation known as "high tracheotomy" should not be taught.[1] He has found "high tracheotomy" to be the chief cause of chronic laryngeal stenosis.

AFTER-CARE OF A TRACHEOTOMIZED PATIENT.

No less important than the operation itself is the constant vigilance required during the first few days following operation.

For the guidance of residents and nurses having the care of patients tracheotomized for any reason whatever, the writer uses a printed sheet containing the following suggestions, taken from Dr. Jackson's book, "Bronchoscopy and Esophagoscopy".

AFTER-CARE OF A TRACHEOTOMIC CASE.

1. Always bear in mind that tracheotomy is not an ultimate object. The ultimate object is to pipe air down into the lungs. Tracheotomy is only a means to that end.

2. Sponge away secretions before they are drawn in.

3. Cover wound with wide, large gauze square, slit so it fits around cannula under the tape holder. Pull off ravelings. Keep wet with 1-10,000 bichlorid solution.

4. Change dressing every hour or oftener.

5. Nurse should remove inner cannula as often as needed and clean it with pipe cleaner before boiling.

6. Outer cannula should be changed every day by the surgeon or long experienced tracheotomy nurse. A pilot should be used and care should be taken not to injure the cut ends of the tracheal cartilage.

7. A sterile, bent probe may be inserted downward in the trachea with both cannulae out to excite cough, if necessary, to expel secretions. An aspirating tube should be used, when necessary.

8. A patient with a properly fitted cannula free of secretions breathes noiselessly. Any sound demands immediate attention.

9. Do not give cough sedatives or narcotics. The cough reflex is the watch dog of the lungs.

10. Bedside tray should contain: Duplicate cannula, Trousseau dilator, dressing forceps, scissors, probe, probe pointed curved bistoury, scalpel, hemostat, sterile vaselin, tape, gauze sponges and gauze squares.

11. Keep mouth clean. Tooth brush. Rinse alcohol 1/10.

12. Sponge away secretion after the cough before drawn in.

13. Remove inner cannula (not outer) as often as needed. Not less often than every hour. Replace immediately. Never boil a cannula until you have thoroughly cleaned it.

14. Obstruction of cannula calling for cleaning indicated by: Blue or ashy color, indrawing at clavicles, sternal notch, epigastrium, noisy breathing (learn sound).

15. Watch for loose parts on cannula.

16. Report at once if food or water leaks through wound (coughing and choking).

17. Never leave a tracheotomized patient unwatched during the first days or weeks, according to case.

18. Remember, Trousseau dilator or hemostat will spread the cannula wound or fistula when cannula is out.

19. Remember, it takes very little to clog the small cannula of a child.

20. Remember, a tracheotomized patient cannot call for help.

Emphasis might be placed on the use of the aspirating cannula for the removal of bronchial and tracheal secretions which may produce obstruction within and below the cannula. The writer prefers the use of a glass aspirating cannula when it can be used with safety as one is enabled to see if the procedure is effective or not and at the same time gain some idea as to the character and quantity of secretion removed.

Just a word, in conclusion, as to permanent removal of intubation tube or tracheotomic cannula. Either of these instruments may be removed as soon as the acute condition for which they were introduced has subsided, providing no permanent stenosis has occurred. All cases from which cannulae have been removed should be kept under close observation for twenty-four hours.

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The Lenox, 13th and Spruce Streets.

LARYNGEAL AND ESOPHAGEAL SEQUELAE OF DIPHTHERIA.*

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Postdiphtheritic sequelae involving the larynx and esophagus present difficult problems to the laryngologist from a standpoint of affording temporary relief to the patient as well as providing a means for effecting an ultimate cure.

Laryngeal Sequelae. These usually consist of a chronic stenosis of the laryngeal lumen and are usually always seen by the laryngologist in the form of a patient wearing an intubation tube or a tracheotomy cannula which cannot be permanently abandoned. In addition, there may occur the panic or the spasmodic forms of stenosis and postdiphtheritic paralysis of the larynx which is extremely rare.

Diphtheria, although a disease which manifests itself by a systemic reaction, is essentially a necrotic process. In these severe laryngeal cases necrotic changes occur not only in the submucosa, but also in the contiguous structures, so that there may result a perichondritis with considerable cartilage loss. With this, mixed infection is the rule, so that one may concurrently find streptococci, staphylococci, pneumococci and other bacteria. There may result excessive hypertrophy about the upper part of the larynx resembling an extensive edematous condition, or there may be marked swelling of the subglottic tissues, the infraglottic type. The latter condition is commonly bilateral, involving the tissues forming the conus elasticus and, encroaching upon the lumen from each side, suggests hypertrophic turbinates.

Extensive fibrotic changes, taking place during the stage of resolution and healing, result in cicatricial stenosis. That this may occur independently of any mechanical manipulation in the larynx itself is undoubtedly true, as it has been the experience of many observers that stenosis follows tracheotomy about as frequently as intubation.

Postintubational Stenosis. In the absence of ulceration it is very probable that no injury to the larynx will result from the wearing of a properly fitted intubation tube. Wearing of an improperly fitted tube, or a tube with sharp edges at its upper extremity sets up a reaction about the upper orifice of the larynx which is described by Jackson[1] as supraglottic hypertrophy. An intubation tube worn

*Read before the Philadelphia Laryngological Society, April 6, 1926.
Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication June 28, 1926.

over a long period of time will become crusted with concretions that produce ulceration. Injury to the larynx during intubation or extubation may produce stenosis.

In the presence of supra- or infraglottic hypertrophic masses the intubation tube undoubtedly acts as a source of irritation. Occurring in the presence of trauma inflicted by changing of the tube under difficult and trying circumstances one cannot expect the larynx to heal normally. Tracheotomy not only puts the larynx at rest, allowing the inflammatory reaction to subside, but also adds very materially to the safety of the patient. A majority of the writers are in accord in this advice. Orton[2] believes that tracheotomy should be performed in chronic diphtheritic stenosis after from four to six weeks if the patient cannot be permanently extubated. This opinion coincides with that held by Jackson, who advises that the question of tracheotomy should be considered after the third week of intubation.

Post-tracheotomic Stenosis. According to Jackson[3] the most frequent cause of chronic laryngeal stenosis is high tracheotomy. Many children have come to the Bronchoscopic Clinic with chronic laryngeal stenosis, the result of high tracheotomy performed as an emergency operation. Tracheotomy as an emergency measure is usually the result of postponing operation too long or attempting to give a general anesthetic. Hasty operation too often means a high incision through the thyroid or cricoid cartilages, hacking of the tracheal cartilages or stabbing of the cricothyroid membrane. Other causes of stenosis are wearing of a cannula of improper size or shape and carelessness in the after-care and neglect of ordinary cleanliness in the wearing of the cannula.

The fibrous tissue in the stenosed larynx may take many forms so that there may be webs running from one part of the larynx to another or a great part of the laryngeal lumen may be obliterated. In the cases associated with perichondritis there may be atresia; usually, however, there remains a minute opening through the larynx.

Treatment of Cicatricial Stenosis. Before deciding on the plan of treatment to be instituted, the larynx should be carefully examined by direct laryngoscopy and bronchoscopy. In the case of a complete stenosis the substenotic portions of the larynx can be readily examined with the aid of the Tucker[4] retrograde laryngoscope.

Obstructing, exuberant granulation tissue and hypertrophic masses should be removed by excision. This method is probably better than removal by the actual cautery. If the intubation tube is still worn and cannot be dispensed with, tracheotomy should be done.

Webs and cicatricial bands may be excised and intubational or, preferably, direct laryngoscopic dilatation carried out while healing takes place. In the cases of necrosis of cartilage with the formation of fungating granulations, thyrotomy may become necessary. In all cicatricial cases the treatment resolves itself into either dilatation by direct laryngoscopy or by laryngostomy, using the Jackson technic. This treatment is prolonged and at times taxes the ingenuity of the laryngologist. Tucker[5] advises the use of a smooth rubber cover over the dilating unit to lessen irritation and promote epithelialization. Baum[6] devised a post attachment to keep the upper part of the laryngostomy opening from closing. After the epithelialized laryngeal lumen has been well established the apparatus is left out and the patient is given a "test period". If there is no tendency exhibited on the part of the laryngeal lumen to contract, the fistula is ready for closure. A sufficiently long period, preferably many months, should elapse, however, before this is undertaken. Closure of the fistula is very satisfactorily accomplished by the Nassau[7] technic.

The ultimate functional result in laryngostomized patients is good. In some cases very satisfactory vocal bands are drawn out from the tissues on the lateral laryngeal walls. In others, phonation is carried on by vibration of the tissues about the upper orifice of the larynx. In one of our cases the arytenoids were drawn forward against the anterior laryngeal wall during phonation, vibrating in that position. The voice is often coarse and harsh, but is of sufficient volume so that it can be well heard.

The following cases, now under treatment at the Bronchoscopic Clinic, Jefferson Hospital, illustrate some of the problems to be considered and the method of treatment to be carried out.

Case 1: F. L., age 6 years, was treated for laryngeal diphtheria two years ago. While in the Municipal Hospital he was intubated, the tube being left out permanently after the fifteenth day. Although the voice was husky and breathing was stridorous at times he seemed to be quite well until two weeks ago, when he developed fever, breathing became noisy and there was dyspnea. This became progressively worse, and on admission to the Bronchoscopic Clinic tracheotomy was immediately performed, using local anesthesia. At bronchoscopy, which was done later, there was found a definite subglottic stenosis. Dilatation by direct laryngoscopy was carried out, using the rounded tracheal bougies. The patient now wears a full rubber cork in the tracheotomy cannula at all times and is ready for decannulation.

Case 2: C. M., age 5 years, had an attack of severe laryngeal diphtheria one year ago. Intubation was necessary and was carried

out for four weeks, when the tube was coughed out. Dyspnea became alarming and tracheotomy was done. Decannulation was attempted, but without success, so the patient was admitted to the Bronchoscopic Clinic about seven months after the original infection. At bronchoscopy there was some fixation of both arytenoids with subglottic stenosis. A size 33, French scale, rounded Jackson tracheal bougie could be passed through the stenotic lumen. Direct laryngoscopic bouginage has been carried out with marked improvement and it is believed that this form of treatment will eventually be followed by complete recovery.

Case 3: A. S., age 2 years, had an attack of severe laryngeal diphtheria at the age of 13 months. Intubation was done to relieve dyspnea. Extubation was immediately followed by marked dyspnea, so a low tracheotomy was performed. On admission to the Bronchoscopic Clinic it was found by laryngoscopy that the laryngeal lumen was completely closed at the level of the vocal cords. Laryngostomy, according to the Jackson technic, was performed ten months ago. Cicatricial tissue was excised and a pack was placed in the cavity. After epithelialization was completed a laryngostomy apparatus was used. The laryngeal lumen is now fully restored and the patient is ready to go on a "test period".

Case 4: J. E., age 3 years, was intubated during an attack of diphtheria fifteen months ago. Following autoextubation, an emergency tracheotomy was done, cutting through the cricoid and part of the thyroid cartilages. Three weeks later a low tracheotomy was performed. When it was found four months later that decannulation was impossible the patient was sent to the Bronchoscopic Clinic. By direct laryngoscopy the upper orifice of the larynx was found swollen shut by two bulging masses. Laryngostomy was done and there was found almost complete destruction of the cricoid and a part of the thyroid cartilage. With the aid of gauze packs and laryngostomic apparatus there now exists a large epithelialized trough extending from the upper laryngeal orifice to the trachea. This is without its normal cartilaginous support and must therefore be maintained in its present state of dilatation until some support can be furnished by either implanting cartilage, bone or other rigid substance, such as ivory.

No one can lay down any positive rules regarding the methods to be utilized except in a general way. Treatment of individual cases must be considered only after a thorough and complete study has been made by direct laryngoscopic and bronchoscopic methods.

Esophageal Sequelae. Involvement of the esophagus as a sequela of diphtheria manifests itself by dysphagia, due either to paralysis or to cicatricial stenosis.

Postdiphtheritic paralysis of the esophagus is of toxic origin and may involve only the inferior constrictor muscle of the pharynx or may occur as a glossolabiopharyngeal paralysis. Occasionally the oblique portion of the inferior constrictor muscle is alone involved, leaving the cricopharyngeal portion intact. The onset is usually insidious, with progressive dysphagia culminating in absolute inability to swallow anything. By mirror examination of the laryngopharynx the pyriform sinuses will be seen filled with secretion (Jackson's sign of esophageal stenosis).

Dysphagia with a history of recent diphtheria should always suggest esophageal paralysis and calls for esophagoscopy. The treatment consists of intubation of the esophagus if no ulceration is present or gastrostomy for feeding purposes and such medical care as may be recommended by the medical attendant.

Postdiphtheritic esophageal stenosis may be the end-result of healing of ulcerated areas in the esophagus, the seat of a diphtheritic infection or of a decubitus ulcer. Councilman, Mallory and Pearce found diphtheritic membrane in the esophagus in twelve cases of their series. In a case reported by Gonin, membrane was found, at necropsy, in the pharynx, esophagus and stomach of a boy, age 11 years, who died of diphtheria. Stupka collected nine cases of cicatricial stenosis following diphtheria of the esophagus.

The diagnosis of stenosis can readily be made by Roentgen ray examination and by esophagoscopy. The treatment consists of dilatation of the esophagus by peroral esophagoscopic bouginage or by retrograde methods if gastrostomy has been required for feeding purposes.

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128 South Tenth Street.

A METHOD OF REMOVING A BEAD FROM A BRONCHUS.

Dr. Simon Jesberg, Los Angeles.

Smooth, round or oval foreign bodies in a bronchus present a peculiar problem. Such objects tend to descend as far as possible, dilating the air tubes and permitting a partial collapse of the lumen above the body. Such a body, if of a hard and smooth character, is almost impossible to engage with any usual grasping instrument, especially if the body is impacted there is insufficient room between it and the walls of the bronchus to permit the passing of an instrument around it. The problem is further complicated by the fact that even the slightest pressure pushes the foreign body deeper and impacts it more tightly.

In the case of a foreign body containing a hole, for an example a bead, the sides of the hole can be used as a grasping surface. If, however, the hole is small, it is difficult to pass into it a probe, or grasping forceps which will be sufficeintly strong to be safe.

The method here described is original so far as I know. It was devised with the aid of Mr. Jacob J. Cantor in the Bronchoscopic Clinic of the Eye and Ear Hospital of Los Angeles, Cal. The device consists of a steel stylet or probe, the distal end of which contains an eye through which a thin rubber band can be threaded. The probe with the stretched rubber band is made just small enough to pass through the hole in the bead. When this is accomplished the tension on the rubber band is released and the expansion of the rubber band locks the bead on the stylet.

Case report: A. J., age 14 months, referred by Dr. J. J. McLoone, of Phoenix, Ariz. April 24, 1926, while playing with sewing basket ornamented with beads, coughed and choked. X-ray showed bead in the trachea. A bronchoscopic examination was made and the bead located, but the operator was unable to engage it with a forceps. There have been attacks of violent coughing and cyanosis; breathing is wheezy and noisy. There has been no fever. Admtited to the Bronchoscopic Clinic of the Eye and Ear Hospital of Los Angeles on May 4, 1926. X-ray showed a large, oval shadow in the right bronchus. After a coughing attack the shadow showed in the upper trachea. Due to danger of impaction in the glottis, an immediate bronchoscopy was performed under butyn analgesia. Subglottic edema was present and a 4 m.m. Jackson tube passed with slight

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Sept. 30, 1926.

difficulty. The bead was easily located in the right bronchus, attempts to engage it in a forceps only succeeded in further impacting it in the bronchus. Four attempts were made to pass a probe into the lumen of the bead; this was not successful. Time: 8 minutes. Five hours later a tracheotomy became necessary in order to relieve dyspnea due to the edema of the glottis.

Mr. Cantor devised the grasping stylet and experimented with many beads, finally selecting a bead estimated to have a lumen the size of that in the bronchus.

May 7, 1926: Lower bronchoscopy, 4 m.m. Jackson tube. It was found that our stylet was too large to enter the lumen of the bead. Smaller stylets were made.

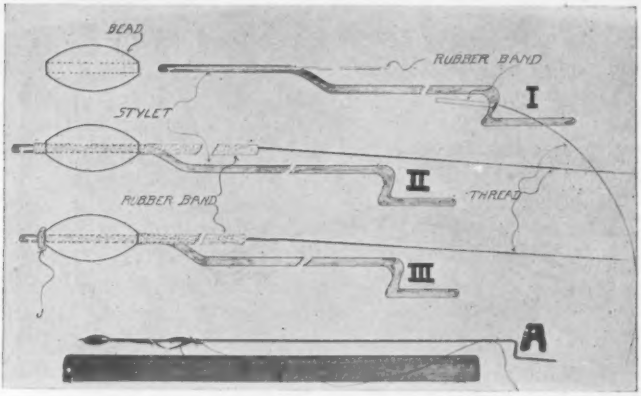

Fig. 1. Stylet with stretched rubber band.

Fig. 2. Distal end of stylet inserted in bead. The expansion of the rubber band locks the bead and stylet together.

Fig. 3. Showing a condition which stometimes takes place. The rubber band "crumpling up" at "J" and serving as an additional lock.

Fig. A. Actual photograph of the stylet and a bead compared with a centimeter scale.

May 9, 1926: Lower bronchoscopy, 4 m.m. Jackson tube. The bead is so embedded in the bronchus that only the lumen is visible; it is difficult to enter the hole in the bead, due to respiratory and cardiac movements. After several attempts it was felt that the stylet was in the lumen, the rubber band was released, and the tube, stylet and bead were removed together. Recovery was uneventful, tracheal tube removed May 13, 1926, and tracheal wound was closed by May 22, 1926.

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